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# PATHOLOGY OF EXPERIMENTAL TRAUMATIC ABSCESS OF THE BRAIN*

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In civil life, bacterial invasion of the brain resulting in the formation of an abscess, occurs relatively infrequently. Such lesions usually follow as a slow extension from a septic process in the neighborhood of the brain and only reach it after erosion of bone and the heavy connective tissues of the meninges. The development may involve weeks or years. It is fortunate that the central nervous system is so well protected from invasion, as the septic injuries of the brain are the most distressing wounds that a surgeon has to handle. During active warfare, on the other hand, the number of such casualties is enormously increased, and this study was begun with the hope of improving the treatment of these cases. Unfortunately, such a program has been but partially carried out and the work here presented represents the histologic control of the experimental traumatic abscesses of the brain.

The first part of the program undertook the study of the repair of aseptic wounds of the brain. This phase of the work was a repetition, on a limited number of animals, of the many similar experiments that have been carried on in other laboratories. Later a large number of animals (thirty-five) were subjected to varying types of infected injuries to discover, if possible, some of the general principles underlying the spread of infection and the factors governing the rate of spread. In every instance the dura was perforated; but the method or the details of infections were varied. Parts of the cortex were scooped out; perforations communicated with the lateral ventricle in some instances, while in others they only slightly injured the most superficial layers of the brain; foreign bodies of bone or metal were introduced into the wound; the skull was fractured in some instances along with the puncture of the central nervous system. In a word — attempts were directed toward reproduction of the wounds received at the battle front.

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EFFECTS OF BRAIN INJURY

It is important to recognize from the outset that an acute traumatic abscess differs in almost every respect from a lesion developing by slow extension. The rapid spread, with its fatal termination in a few days, as well as its healing by the formation of a connective tissue scar, presents an entirely different picture from the slowly growing process with a distinct tendency toward sequestration by forming a thick connective tissue capsule. The reasons for such differences are not to be sought in the kind of infection, the virulence of the organism, the kind of defensive mechanism employed by the affected part, or its anatomic position, but rather in the effect of the *injury* itself. Injuries involving the central nervous system at once cause a profound alteration in the normal circulatory and drainage mechanisms of the brain. The expression of such changes is swelling, or edema, and we have the beginning of a vicious circle, which takes but hours to be set in full motion. Long before anything which could be looked on as an adequate interference with bacterial growth has been instituted, the tissue is made more susceptible by anemia and edema. It is the surgeon's chief problem when operating on the slow eroding abscess of extension, to avoid exciting by trauma the phenomena of swelling and edema, and thus producing a rapidly fatal extension of the process.

The literature on brain abscess is clouded by the use of terms of which the exact meaning is not always clear. The susceptibility of the highly specialized nervous tissue to modifications of the vascular system, which might be inconsiderable in other parts of the body, opens up an easy field for softening and necrosis. It is to these processes that much attention was given in the older literature with efforts to establish their relation as fore-stadia to inflammation. These softenings were usually included under three heads — red, white, and yellow, according to the color seen at gross necropsy. The first two depend on the relative amounts of blood in the outer zone of the abscess; the more common red softening being the result of extravasated erythrocytes and vascular dilatation; while the white softening follows the anemic conditions and is more often associated with thrombosis of a vessel. The yellow, sometimes brown, softening is an older stage of the red softening and results from the breaking up of red cells into various oxidation products of hemoglobin.

The term "encephalitis" itself seems to be simple and straightforward enough, yet it suffers confusion with "inflammation," particularly within the central nervous system where vascular reactions are so greatly modified. It is repeatedly discussed in its relationship to red softening in a way that seems to cloud rather than clear the con-

ceptions. The cause of the confusion is unquestionably attributable to the fact that our knowledge of the encephalitides is mainly classified after study of clinical history and resting very little on pathologic findings under the microscope. Our knowledge of the actions of toxins on the nervous system is very incomplete, but it is here that the key to many of our difficulties lies. The relation of abscess to encephalitis is described by Oppenheim,[1] as follows:

> Es gibt Encephalitiden, die mit dem Hirnabscess nichts zu thun haben, nicht in denselben übergehen. Es gibt ferner eine haemorrhagische Encephalitis, die sich mit einer suppurativen verbinden, respective in dieselbe übergehen kann (durch secundaere Infection). Dies gilt z. B., für die traumatische. Der Hirnabscess entsteht wahrscheinlich meistens in der Weise, dass von vorneherein zur Eiterbildung kommt; jedenfalls ist es nicht bewiesen, dass dem Stadium der Suppuration ein anderes voraus zu gehen braucht, in welchem das anatomische Bild sich mit dem der "rothen Erweichung" deckt.

The use of these terms has been purposely avoided in an attempt to introduce as little confusion as possible into this paper.

Experimental work on the central nervous system has been largely directed toward the simple repair of the brain lesions under aseptic conditions with a view toward establishing the part played by the various cellular elements — fixed and foreign — in the reestablishment of an equilibrium. The earliest researches were concerned with the ability of the nervous tissue to regenerate. The ganglion cells, for the most part, play a passive rôle and show but a feeble effort to replace destroyed elements. Their nuclei may show a preparation for mitotic division, but there is little evidence that this division is ever completed normally. The same holds true for the regeneration of the axone, within the central nervous system, when once the continuity of the latter is broken, nothing comparable to the replacement in a peripheral nerve ever occurs. The neuroglia cells are found to play a very important part both in inflammation and repair of the brain.

The best work on simple wounds of cortex was carried out in Ziegler's laboratory by Tschistowitsch.[2] To prevent the inflammatory factors from complicating the histologic picture of repair was his chief aim, and his choice of foreign body (small celloidin tubes kept sterile in alcohol) seems most fortunate, in that it produced practically no tissue reaction in the brain. He found that the nervous elements of the neighborhood made no attempt to reproduce the loss. The glia

1. Oppenheim, H.: Die Encephalitis und der Hirnabscess, Nothnagel's Specielle Pathologie u. Therapie, Wien. **9**: 1903.

2. Tschistowitsch, Th.: Ueber die Heilung aseptischer traumatischer Gehirnverletzungen, Ziegler's Beiträge **23**:321 (2 plates), 1898.

cells swelled up, divided actively — probably as a result of the change in nutrition. Tschistowitsch has no proof that they add to the host of phagocytic cells which digest and remove the destroyed tissue, but he does not conceive it impossible. He believes the phagocytic cells to be derived from connective tissue cells of the pia and from the adventitia of the penetrating vessels, partly from endothelial lining of blood vessels and leukocytes. The substitution of lost parts is made almost entirely by connective tissue. Within a few days the young fibroblasts derived from the pia mater may be seen wandering into the wound edges with their long axes parallel to the axis of the lesion. Side by side grow new vessels. The connective tissue growth was much less in wounds produced by introduction of celloidin tubes. The glia takes no part in the formation of the cicatrix which replaces the destroyed elements, but merely confines itself to the elaboration of a secondary zone of sclerosis around the scar. The sclerosis varies in amount with the duration of the original stimulus.

The activity of the neuroglia in inflammation as well as in normal conditions has been elaborated on by Alzheimer.[3] He postulates two kinds of neuroglia cells, indistinguishable from one another, which exist as a normal supporting reticulum. Those of the first type are essentially part of the fibrillar network and they are solely occupied with the maintenance of this network. A second variety may easily free themselves and become ameboid. This group, under the stress of great degenerative processes, takes on special forms, assimilates degenerated products, and carries them to the perivascular spaces. Thus they may contribute to the large phagocytic mononuclears seen in close association with the brain abscesses.

### RELATION OF MACROPHAGE TO ABSCESS

The most striking characteristic of destruction of the nervous tissue is the appearance of these large mononuclears. In 1841, Gluge[4] called attention to *Körnchenkugeln* as a diagnostic point in inflammations of the central nervous system. These were collections of *Körnchenzellen*, about which all subsequent discussions concerning inflammatory processes in the brains revolved for the following half century. Virchow[5] described an "encephalitis neonatorum" in which he regarded

3. Alzheimer, Alois: Beiträge zur Kenntnis der pathologischen Neuroglia und ihrer Beziehungen zu dem Abbauvorgängen im Nervengewebe, Histol. u. Histopathol. Arb. über Grosshirnrinde (Nissl), Jena **3**: 1910.

4. Gluge, G.: Experimente über Encephalitis, Abhandlung z. Physiol. u. Path. No. 2, Jena, 1841, p. 13.

5. Virchow, R.: Kongenitale Encephalitis und Myelitis, Virchow's Arch. **38**:129, 1867.

the *Körnchenzellen* as a product of a fatty metamorphosis of neuroglia cells. Hayem[6] looked over twelve supposedly normal new-born brains and found granular cells (*Körnchenzellen*) which he took to be the normal appearance at that period of growth. In 1871, Jastrowitz[7] demonstrated their presence in children's brains unquestionably normal because they were dead only as a result of accident at birth. In addition he proved that there was an apparent irregularity of distribution. He noted the greater number of these cells to occur in these areas where fibers were developing more rapidly than in other regions of the brain — "besonders stürmischen Entwicklungstypus." He thought them of glial origin. Boll[8] identified them in normal chick embryos at from twenty to twenty-one days of incubation, and found them extremely ameboid on the warm stage. He believed that they brought fat for the deposition of myelin on the axis cylinder. Flechsig[9] completely correlated their distribution with the time of myelinization of the fiber tracts, yet he was unwilling to commit himself as to their origin, but inclined toward a view that they were glial. The microchemical work of Wlassak[10] solved the discrepancies which gave rise to so many diverse interpretations. On the one hand, the followers of Virchow regarded these granular cells as degenerative in character and consequently to be viewed as pathognomonic of inflammation. On the other hand, Jastrowitz and his followers believed their occurrence was a normal physiologic event, especially when associated with early development. Chemical analysis proved both of their views correct although the controversy had lasted for many years. Granules in cells, then, could be lecithin, protagon, albumin, decomposition products of hemoglobin, fat, and so on. With this conception, the cells previously grouped as "Körnchenzellen" have been variously subdivided, according to the conception of the author as to origin, morphology, or physiologic activity. The more recent literature has become more and more interested in the function and activities of cells and less with exact morphology. Such interpretations are open to the possibilities of greater mistakes, but also allow a more intelligent effort toward control of cellular reactions in inflammation and repair.

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6. Hayem, G.: Etudes sur les diverses formes d'encefalite, Paris, 1868.

7. Jastrowitz, M.: Encephalitis und Myelitis des ersten Kindesalters, Arch. f. Psychiat. **2**:389, 1870; **3**:162, 1872.

8. Boll, Fr.: Die Histologie und die Histiogenese der nervösen Zentralorgane, Arch. f. Psychiat. **4**:1, 1874.

9. Flechsig, P.: Die Leitungsbahnen im Gehirn und Ruckenmark des Menschen, Leipzig, 1876.

10. Wlassak, W.: Die Herkunft des Myelins, Arch. f. Entwicklungsmechanik **6**:453, 1898.

Among the writers of the last decade are found Nissl[11] and Mertzbacher[12] who have attempted to read activity into the processes of destruction and repair, each from slightly different points of view. The experimental work of Nissl has served to emphasize the "Gitterzell" as a pure and simple phagocyte whose chief rôle is the restoration of order after a destructive process has set in. The granular cells should no longer be looked on as a specific element in inflammation, for they are quite often the cells wholly concerned with reparative process. They appear in every situation where necrotic tissue-masses (foreign bodies) must be removed. If one chances on these cells at some distance from the lesion within the perivascular spaces, more than likely they have come from the focus of infection, laden with waste products and are making their way into the channels of tissue-drainage.

Mertzbacher wishes to reclassify these cells seen in inflammation into a physiologic group and ignore their origin entirely. His term "Abräumzell" suggests a purposeful activity and includes both "Aufbauzell" and "Abbauzell." In this sense it is better than the physiologic term "Macrophage," but the latter is probably doomed to stay because of its priority. The term "Macrophage," proposed by Metschnikoff, refers only to the power of the cells to phagocytize large elements. The functional division made by Mertzbacher was between physiologic and pathologic cells — the normal physiologic "Körnchenzell" of the embryonic central nervous system (clearly an "Aufbauzell") belongs to the group of cells known as "Abräumzell of the adult.

Friedmann[13] published many papers on his experiments on encephalitis, emphasizing the importance of the epithelioid cells. His conceptions and observations seem in no way to have contributed to the solution of the problem.

### THE WALLING-OFF PROCESS

The question of greatest interest to the surgeon has always been the walling-off process. Where the invasion is slow, the fibroblasts lay down a very efficient barrier of connective tissue ahead of the infection. The time necessary to bring about the protective barrier has been variously estimated, and the lower limits necessary for its formation have been the subject of much discussion. The knowledge

11. Nissl, F.: Zur Histopathologie der paralytischen Rindenerkrankung, Histol. u. Histopathol. Arb. über Grosshirnrinde **1**:315, Jena, 1904.

12. Mertzbacher, L.: Untersuchungen über die Morphologie und Biologie der Abräumzellen im Zentralnervensystem, Histol. u. Histopathol. Arb. über Grosshirnrinde (Nissl) **3**:1, Jena (7 plates), 1909.

13. Friedmann, M.: Encephalitis und Hirnabscess, Handbuch der Pathol. Anat. des Nervensystems (Flatau-Jacobsohn) Berlin **1**:494, 1904.

gained from aseptic wounds helps us but little. There the young fibroblasts may be seen on the fifth to sixth day, taking active part in the repair, but in no sense can they be looked on as capable of resisting the spread of an infection. The differences of opinion are largely the results of inability to absolutely demonstrate true connective tissue fibrils. Preceding the actual fibril formation, a collagenous material is laid down, and this takes a progressively deeper stain, similar in character to the finished connective tissue.

This process of repair in a sterile wound is in no way comparable to the formation of a protective wall in an abscess. The latter forms in the intermediary zone between the living and dead tissues, attacked on one side by the toxins and supported on the other by a very much impaired circulation. The fluids bathing the young fibroblasts which wander very early into the outer zone of the abscess are certainly very different from those present in a sterile destruction. It is generally conceded that an adequate connective tissue barrier forms very slowly. Observers have made various estimates from clinical history, namely, Lebert, from 3 to 4 weeks; Schott, from 7 to 8 weeks; R. Meyer, 7 weeks; Gull, 10 weeks; Huguenin, 53 days a thin membrane, 83 days a thick resistant wall.

The lowest estimate in these figures shows the utter hopelessness of waiting for such a fibrous barrier to help us in treating a traumatic infection of the central nervous system. Furthermore, it must be remembered that, were its formation possible, it would be of doubtful value when one considers that the completely encapsulated process slowly encroaches on the healthy tissue owing to toxic products seeping through the capsule, through pressure, or through vascular changes. It may be repeated that the traumatic abscess is something different from the encapsulated variety and by careless handling the latter may be transformed into the former. The infection in the brain spreads so rapidly after an injury that there is no time for the interposition of a connective tissue barrier, and a rational treatment can never hope to receive aid from such a slow and really ineffectual mechanism.

## METHODS EMPLOYED IN EXPERIMENTAL PRODUCTION OF TRAUMATIC ABSCESSES

It was early discovered that an abscess in the brain of a cat developed with great uncertainty unless a positive—what might even be called a massive dose of bacteria was introduced into the wound. Simple puncture wounds often healed *per primam* although no efforts were made to sterilize the instruments or tract. An animal's resistance to infection, even after rupture of the dura, was found to be quite remarkable. There seemed to be little tendency

for organisms to grow down into the brain from the skin, or for the few carried in with the instruments, to gain a foothold. In order to inaugurate an infection, an injection of from 0.1 to 0.3 c.c. of bouillon culture or pus in varying dilutions was made into a wound. The ordinary pyogenic organisms cultured from human infections were found to be very feeble in virulence. Pure cultures of bacteria obtained from abscesses in cats proved to be more efficacious. The most rapidly fatal lesions were produced by taking pus from a cortical abscess of one cat and injecting it directly into the brain of another. Some of the animals treated in this way survived less than twenty-four hours. Not uncommonly a spread along the whole track of the wound instituted a cellulitis within the scalp. Such secondary complications were treated by evacuation as soon as they developed, but at times the subcutaneous infection would spread over the entire head and as far forward as the eyes. The cats within this series were not all normal when the experiments were started, often having been used for other observations on the central nervous system. Such a past history often changed the course of the abscess as well as the cellular reaction of the host.

A nail of given length was sharpened for making perforating brain injuries and a known weight was put behind it; this was then allowed to fall a definite height. The method was quickly abandoned because of the uncertainty of getting a standard wound. The principal difficulty lay in the rotundity of the cranium. Blows administered in this manner tended to be deflected so that considerable subcutaneous trauma might be inflicted without perforating the skull. In one experiment a piece of infected copper wire was forced into the brain substance through the nail hole. Another method employed was to remove a cylinder of the skin, skull, dura and brain, and to place infected foreign bodies in the defect. Such a procedure at times reached the lateral ventricle, more often the infection did not start within the ventricular system. A trephine was used for the skull and a cork borer to remove soft parts.

The greater number of experiments were so made that there was no room for expansion of the brain. A bluntly sharpened instrument, 1 mm. in diameter and provided with a shoulder so that it could only be inserted to a depth of 6 mm. from the surface of the skin, was used to make an opening for a hypodermic needle. Such wounds caused little or no trauma to the subcutaneous tissues and were just sufficient to penetrate the outer zones of the cortex. Rarely this struck a sulcus and the syringe needle actually ended in the subarachnoid space without penetrating the cortex. At necropsy such failures were indicated by a purulent meningitis without involvement of the brain. The quantity of diluted pus or bouillon culture used was sufficient to cause a localized rupture of the tissues accompanied by slight hemorrhage. In every case an initial spread of the injection mass into the subarachnoid spaces was unavoidable.

A few attempts were made to completely localize the infection to the point of inoculation by injecting the septic material into the exposed cortex through a trephine hole and immediately sterilizing the surface. The results of such a procedure were very disappointing as they were just as susceptible to a meningeal spread as were animals treated similarly without sterilization. Attempts were made to cure the experimental abscesses either by trephining and draining, or by trephining and irrigation. The number of attempts was

so small that no conclusions could be drawn. Actually, the number of spontaneous cures was greater than the number following attempted therapy.

## CLINICAL HISTORY

Little importance can be attached to this part of the work in animals. Often, when the virulence of the infecting agent was low, the animal behaved as if normal for a day or two. On the other hand, with more virulent strains injected with pus, the protocols show that the cats were sick immediately after the experimental injection. Following concussion or cortical ablation the animals were dazed or showed little desire to move or eat; at such times there was a definite weakness in the hind legs but no actual paralysis. After considerable trauma they might be unable to walk or crawl, but these reactions should not be considered in any differential diagnosis concerning the type of lesion. The history of the present illness would be enough to account for the reaction. No attempt was made to record temperatures as there is so much variation under the condition of experimentation. After simple perforation the element of trauma was minimal, and the record of "quiet" or "less active than normal" would be the only signs of disturbance. The discharge of pus from the wound gave absolutely no clue as to the process developing within the cortex. As a matter of fact, this hole in the bone was very quickly sealed by granulation tissue and the subcutaneous reactions had no effect on the cortical infection. Very few of the cats were sacrificed but were allowed to die spontaneously. The animals included in the end of the series were often found dead in less than twenty-four hours after inoculation.

## PREPARATION OF MATERIAL

The animals were embalmed as soon as possible after death by injection of 10 per cent. formalin through the aorta. A block including the skull and cervical vertebrae was then suspended in the same fluid until ready for study. The relations of the meninges to the bone were studied and gross macroscopic descriptions were recorded and the tissues were embedded in celloidin. Hematoxylin and eosin, toluidin blue, Van Gieson, and Mallory's connective tissue stain were applied to the sections. It is evident that such technic precludes a study of the finer cellular changes in the nervous system, but it preserves the intracranial topography and does not interfere with observations of the cellular reactions.

## MACROSCOPIC APPEARANCE OF BRAIN ABSCESSES

The central nervous system, as usually seen at necropsy, gives little exact knowledge of the relations obtaining at the time of death. Dislocations and changes in shape due to pressure are largely lost when the soft brain is taken from the skull. By injecting formalin into the aorta at death and fixing thoroughly by immersion, all of the gross form relations are preserved. The changes in the size, shape and position of the ventricles are maintained faithfully and any distention of the leptomeninges by fluid-accumulations is undisturbed. Unless such fluid in the subarachnoid space is coagulated by fixation,

its appearances of distention are largely lost if the meninges are not hardened in situ.

The most striking change produced by these traumatic abscesses was the increased tenseness of the brain. This showed itself most markedly by a herniation of the subjacent nervous tissue through a defect in the dura and skull. Whole gyri were capable of slipping out when the hole was large enough, taking with them a fairly intact blood supply. Such protrusions rapidly underwent necrosis in the central portions and were sloughed off. The most rapid expulsion of nervous tissue followed the simultaneous development of a subdural abscess away from the dural defect, and this greatly added to the fungus which developed as a result of increase in size of the brain itself. Palpation of such a brain revealed an unusual tenseness, and any splitting of the dura due to careless removal allowed the brain to bulge out. The evidence of swelling might extend down the cord where the markings of the intervertebral spaces could be seen in outline. On the dorsal surface of the cord these segmental corrugations were often great enough to be felt by running the fingers up and down the unopened dura. The result of the swelling was to partially obliterate the sulci of the brain so that they appeared as mere lines when viewed through the dura. Such conditions might still exist at the end of a week. The effects of swelling were usually more marked on the affected side, but they might involve both cerebral hemispheres equally. The macroscopic evidences of meningeal infection, hyperemia and petechiae, clouding of the normal markings and heavy purulent exudate, showed considerable variation. In six of the experiments, no evidences of an inflammation spreading beyond the point of injury could be demonstrated macroscopically. In seven instances, there was a progressive advance of the process over the injured hemisphere with the formation of a heavy purulent exudate which completely obliterated the brain markings on the affected side. There was a distinct tendency to stop at the midline along the falx cerebri (Fig. 5), and to spare the temporal pole and the occipital lobe. Another peculiarity of distribution, in two experiments, was a normal looking cerebrum surrounding the injury with no macroscopic evidences of meningitis over the brain; the cord, medulla and cerebellum, on the other hand, were totally obscured by a thick layer of yellowish exudate. In these instances either by direct injection into, or subsequent invasion of the lateral ventricle, the ventricular system became the pathway for an extension of the infection to the natural outlets to the subarachnoid space — the foramina of Magendie and Luschka. At these points the process attacked the meninges spreading over the hind brain and

down the cord. In fifteen of the thirty-five animals, a generalized meningitis was present at death. Where only a small opening existed in the skull and there was no chance for the nervous tissue to herniate, the defect was filled with a plug of coagulated debris which quickly began to organize. Some of the injuries were very close to the mid-line and an opening was made into the large vein emptying into the sagittal sinus. This was followed by a hemorrhage which almost completely covered the cerebral hemisphere, extending to the base of the brain and out along the rhinencephalon.

On section, the gross specimen showed remarkable changes in brain-bulk, which brought about extensive dislocation of the entire brain, especially where the lesion developed rapidly within the normal confines of the skull. The affected hemisphere might occupy one-third again as much space as the unaffected one, enlarging at the expense of the latter (Fig. 10). This encroachment of the injured side was greatest at a point midway between the vault and the base of the skull. At these fixed points the nervous tissue was anchored by the meningeal vessels, and had no opportunity to be displaced. The result of this was a curved midline with the convexity looking toward the unaffected side. The transverse section through the lesion showed the greatest changes, but the entire affected hemisphere might bulge beyond the normal limits (Figs. 10, 11). As long as the brain herniated, as in a decompression (Fig. 19), the normal appearance of the mid-line was unaffected. Here the local increase of brain-bulk took the line of least resistance and instead of compressing healthy brain tissue, release was afforded by protrusion from the skull.

Another effect of increase in size of the brain was the encroachment on the spaces ordinarily filled with cerebrospinal fluid. Such a condition arose in most of the experiments included in this series, because the abscesses developed within the intact skull. The gyri were forced together and the normal subarachnoid tissue between them was compressed until it appeared as a mere linear slit. The normal, rounded gyrus was flattened out against the dura and the contour approached a straight line with the sulci meeting it at right angles. It is conspicuously shown in Figures 11, 16 and 18. The reactions of the ventricular system were very definite. They were complicated by infection in eighteen instances, of which three were unquestionably inoculated at the very beginning of the experiment, whereas fifteen were infected by extension of the process into it. Whether invaded or not, there was present, in nearly all of the experiments, a tendency toward the production of a slight grade of hydrocephalus (Figs. 8, 10

and 11). If the ventricle was perforated by the original injury, the entire ventricular system rapidly dilated with pus (Fig. 18) forming a pyocephalus of moderate grade. Where the original abscess eroded its way into the ventricle, the latter was soon filled with a purulent exudate on the affected side. The contralateral ventricle was likewise distended, but as a rule the character of the exudate was clear and coagulated with fixation (Fig. 10). The entire ventricular system may be thus dilated. No interference to the normal outflow of cerebrospinal fluid could be discovered at the foramina of the fourth ventricle. An abscess developing within a confined space compressed the lateral ventricle directly beneath; if the brain was able to herniate from the skull, the ipsilateral ventricle was distended at that point. On the whole, the contralateral ventricle is more widely distended than the ipsilateral.

The bacterial invasion of the brain substance has shown marked variations in the rapidity of its spread, but an almost universal predilection to follow the fiber tracts rather than spread in the cortical gray matter. The result of such a mode of spread was the production of a pear-shaped lesion (in the early stages), the narrow neck of the abscess being located at the point of destruction of the cortex. At times this extended around neighboring sulci by picking out the fibers and from there it more slowly eroded its way through the cortex to the meninges. In some of the most virulent infections an abscess 2 cm. in diameter was found within twenty-four hours, being large and toxic enough to kill the animal. The core of the abscess quickly became granular and opaque. This necrotic center was surrounded by a more translucent gray zone while the latter, in turn, was hemmed in by a zone of punctate hemorrhages. The tendency toward ecchymosis was absent in very few instances, but often extended for a considerable distance beyond the actual necrotic process. By carefully picking (with a delicate instrument) the edges of the process it was impossible to demonstrate a wall of any tougher consistence than the normal nervous tissue itself; the edges everywhere were quite friable.

The central core was usually granular and somewhat inspissated, but in some instances it was represented by a clear gelatinous exudate (Fig. 12) which projected slightly from the plane of section. The walls of such a cavity were smooth. In one experiment there was present a gas-forming organism which produced a considerable gas filled cavity in the center of the abscess. As a rule, however, the abscess consisted of a necrotic core located for the most part in the white matter of the cerebrum and surrounded by a zone of small ecchymoses.

Marked change in color took place in the macroscopic appearance of the fiber tracts. It appeared first as a more translucent core developing within the center of the fiber tract near the abscess and radiating for somé distance on the affected side. In the affected area, a thin fringe of white matter adjacent to the cortical gray preserved its pearly white appearance and served to set off the central part by contrast (Fig. 8). In most of the experiments this change was fairly well limited to the affected side, but occasionally it extended into the opposite hemisphere along the corpus callosum. Gradually this gray translucence spread to the outer limits of the fiber tract and the distinction between gray and white became almost obliterated (Fig. 12). It seemed to bear some relation to the pressure, inasmuch as it was less well developed in those cases in which a decompression had allowed the brain to expand.

There was a marked tendency for the arachnoid to be split away from the dura by a rapidly developing meningeal abscess. Even though a hole exists in the skull (Fig. 19), this collection of pus cannot evacuate itself because the expanding nervous tissue forms an efficient plug for the outlet. In twelve instances a considerable collection of subdural pus was present. A meningeal abscess of remarkable size followed an attempt at osteoplastic repair of the skull after the cortical abscess had been completely healed (Fig. 8). Such a meningeal abscess developed with remarkable rapidity and at times invaded the opposite hemisphere by extending down one side of the falx and back up the other. These abscesses have shown no tendency to invade the cortex, although they must exert a considerable pressure upon it.

### MICROSCOPIC APPEARANCE OF BRAIN ABSCESSES

Under the low power of the microscope, the typical brain abscess in these experiments presented three well marked zones — a central core of necrosis, a surrounding fringe of leukocytes and a zone of vascular engorgement and hemorrhage. In the first two, all semblance to structural characteristics common to the tissue was obliterated, while the last, called by many writers "the necrobiotic zone," still maintained characteristic appearances and staining reactions, and represented the transition between the dead and the normal living tissue. The destroyed center took the eosin stain very poorly and the hematoxylin not at all, except for patches of dense blue colonies of bacteria. The densely staining bacterial masses were found to be thickest in the periphery of the necrotic zone, just inside the cellular layer. Often within the necrotic zone surrounding a blood vessel, appeared small islands of tissue which had not been totally reduced to a homogeneous

appearance. The "burnt-out" character of the central core may be extensively developed within a few hours if the virulence of the organism is great. Where the process developed more slowly, the abscess had a laminated appearance (Fig. 14), as the cellular zone progressively died and was in turn encircled by a new layer of exudative cells. The width of the second or middle zone varied considerably. In a rapidly advancing process this layer was well developed and the cells infiltrated the tissues in enormous numbers. At such a time the outer limiting edge was comparatively smooth (Fig. 13). Apparently, the resistance of the tissue to invasion showed little local variation, and the process swept through the brain without the usual degenerative changes occurring in front of it. Beyond the leukocytic zone, as seen under the low magnification, there were changes referable especially to the vascular system. Red blood corpuscles had extravasated in quantities sufficient to obscure the texture of the nervous tissue. Serous infiltration may give it a loose watery appearance. Radiating from the abscess, the perivascular spaces were often packed with nucleated cells for a considerable distance (Fig. 3).

Low magnification demonstrated most clearly the relative resistance of the different regions of the cerebrum to infection. The fiber-tracts were involved more rapidly and extensively than the gray matter. The cortical gray itself showed differences in the susceptibility toward destructive processes. Thus the molecular zone might survive, although for a considerable distance (Fig. 16), the ganglionic layers beneath and the meninges above showed complete destruction. Beyond the outer zone of hemorrhage and vascular change, the fiber tracts are usually much lighter in density of eosin stain than normal fibers. The initial stages of the translucent gray appearance, developing in the fiber tracts concomitantly with the increase in intracranial pressure, could be made out only under very low power of the microscope. This change was manifested by a slightly lighter stain with eosin when compared with the normal fiber bundles of the unaffected hemisphere. Under the high powers the differentiation between the affected and unaffected sides was impossible until the process had progressed for a considerable period. The subdural location of the meningeal abscess is well brought out in Figure 14. Owing to the shrinkage in embedding, an artificial separation has been produced between the abscess and the pia-arachnoid. The latter was heavily infiltrated with cells, but was distended very little more than normal.

A minute study of the central necrotic zone, during the early stages, revealed the shadows of exudative cells, faintly staining with eosin.

Rarely was it possible to recognize any of the original structures excepting the larger blood vessels. By crushing bits of this necrotic center on a slide and staining it with weak aqueous methylene blue, a very good idea of the composition of the mass could be gained. It was there easy to pick out the structural elements, ganglion cells, glia cells, polymorphonuclear and mononuclear leukocytes making up the dead core. Almost every cell contained organisms, and it was striking that the bacteria were for the most part intracellular. At times the cells and their contents took no stain whatsoever, appearing as a simple envelop with refractile inclusions. Again, the cellular envelop contained from ten to twenty stained organisms, but otherwise was colorless. Still other cells have been found in which the blue-stained nucleus could be made out in addition to the bacteria. The intracellular position of the organisms could not be seen in the cleared and stained sections, partly as a result of the thickness of the sections, with a resultant superposition of elements, and partly because the clearing made cell boundaries undistinguishable.

The distribution of organisms within the necrotic center was fairly uniform, but as one neared the zone of cellular exudate the number and density were greatly increased so as to form a bluish zone inside the border of living cells. Figures 7 and 15 show very clearly these bacterial masses flourishing on the very rim of the necrosis. In Figure 15 one gets the impression of an actual radial advance of these masses into the leukocytic zone and a trail of destruction in their wake. The organisms were usually mixed where pus had been the original infecting agent, but most frequently in these experiments the destructive organism was a streptococcus. This was the organism which was found massed behind the leukocytic zone. The isolated islands appearing within the necrotic area were built around the larger blood vessels and were composed of collections of exudative cells or at times mainly red blood corpuscles in a fairly good state of preservation.

The "burned-out" appearance was rather extensively spread when the lesion developed rapidly and then could be seen the pale shadows of the exudate over wide areas. In a short time this was replaced by an amorphous granular detritus in which a few bacteria could still be seen. The liquefaction of the debris was a rather rare phenomenon, only occurring in two instances. The failure to find this more frequently might be referred to short period of survival of the animals in most of the experiments. Here it was not unlikely that the liquid was a coagulated exudate of a fluid free from cells. Microscopically,

such an area appeared perfectly homogeneous, eosin-staining, and with no indication of any formed elements or bits of debris.

Surrounding the necrotic core was a zone of cells which was made up of a preponderance of polymorphonuclear leukocytes in the early stages. Very rarely the mononuclear infiltrating cells outnumbered them; then no accountable reason could be found. These small cells were the derivatives of the circulating blood and formed a closely packed boundary to the abscess. The outer limits of this zone was usually beyond the extension of demonstrable bacteria, sometimes, however, it was coincident with their growth, and very rarely the bacteria spread beyond their outer limits into tissue, recognizable structurally as nervous tissue. Sometimes, in localized areas, the bacteria grew beyond the infiltrated zone into the perivascular sheaths with no cellular reaction (Fig. 13). The more usual conditions are illustrated in Figures 7 and 15, where the leukocytes interpose themselves between the bacteria and the surrounding nervous tissue.

Enveloping the entire process was a shell of tissue in which the vascular changes were the predominant features. All the vessels were engorged with red blood cells, which in many places have escaped into the tissues. The ground-work was also infiltrated with a serous exudate, often coagulated and containing spicules of fibrin. There were many isolated leukocytes scattered in this area, but in general they were found in greatest numbers in the perivascular spaces (Robin) radiating from the edges of the abscess. Often the smaller vascular branches were so densely surrounded by these wandering cells that the lumen appeared compressed (Fig. 3). At some distance from the lesion they were confined absolutely to the perivascular spaces, but, as the edge of the leukocytic zone was approached, they invaded the tissue in increasingly greater numbers. The distention of the tissue elements by exudate was quickly followed by swelling up of the glia cells. These are normally inconspicuous with little protoplasm about the nucleus; in these cerebral infections after a few days they appeared more rounded and at times in the process of division by mitosis. The most striking phenomenon was the escape of erythrocytes from the vessels; this happened in varying degree in all of the experimental abscesses in this series. At times the ecchymosis was great enough to form an almost unbroken ring of free erythrocytes around the zone of exudative cells (Fig. 6) even extending for a considerable distance into what might be viewed as otherwise normal tissue. At other times they appeared as small punctate hemorrhages scattered around the periphery of the lesion. Such collections of red

blood cells usually formed a locus of increased resistance toward bacterial involvement. These islands of red cells indented the advancing edge of the process (Fig. 7) and only succumbed after being entirely surrounded by the infection. The ganglion cells in this outer zone showed the classic signs of degeneration — clumping of the Nissl bodies, chromatolysis, shrinking of the nucleus and disappearance of the cell. After several days this zone was invaded by young fibroblasts, most of which were derivatives of the pia-arachnoid and of the adventitia of the neighboring blood vessels. Elongated typical cells could be found developing from the meninges at the edges of the wound, and from this point they extended into the depths within the zone of necrobiosis. The adventitial sheath of the blood vessels in the neighborhood of the lesion also proliferated by mitosis and furnished fibroblasts which extended into the outer limits of the abscess. In none of the specimens was there the slightest evidence of an orderly arrangement such as might be looked on as a foundation for a capsule — nothing which could be interpreted as an early attempt on the part of the fibroblasts to interpose a barrier against the spread of the infection.

For the first two days in the development of the abscess, the infiltrating cells were composed almost entirely of leukocytes. After that the necrobiotic zone was invaded by increasing numbers of large mononuclears, known as "macrophages," which collect where the extravasation of blood is greatest and among the fiber tracts that were undergoing secondary degeneration. These cells were most conspicuous for their size, actively engulfing the products of tissue destruction until they reached enormous proportions. Such cells are so well known that an exact description of their appearance is superfluous. At times they were gathered in close formation occupying entire microscopic fields, their pale reticulated bodies and eccentric nuclei being in close apposition to one another. The activities of these cells in removing a blood clot are truly remarkable. This is well illustrated in Figure 16, a photograph of a brain section into which a button of bone was forced and a dense blood clot resulted. The lower central portion of the mass has been almost entirely removed by activity of these cells. Most of these are derived from the pia-arachnoid, adventitia of blood vessels, endothelium and neuroglia cells, where they could be seen to develop from the fixed tissue cells and to take on the characteristic lattice-work structure, typical of the protoplasm of the macrophage. When found among the degenerating fiber tracts their bodies were loaded with droplets that stained with osmic acid. At times they formed an almost

unbroken ring around the abscess just outside of the leukocytic zone. As the process spread they were included within the middle zone, but one gathered the impression that they were not associated with the active process in the same way that the exudative cells of the blood were concerned. Rarely these large phagocytes had entered the outer zone of the abscess in considerable numbers within forty-eight hours. Some of these animals had been previously used in other experiments and, in all probability, a considerable number of the macrophages were in the meninges at the time of the production of the abscess.

The traumatic brain abscess has been found to be occasionally accompanied by a considerable amount of transudate extending throughout the lesion. The zonular arrangement just described was then more or less obliterated. The pattern became a sort of mosaic of dead tissues, islands of exudative cells, homogeneous patches of eosin-staining material, and masses of red blood corpuscles. Throughout were crystals of fibrin embedded in a clear matrix, or woven among a loose collection of leukocytes. In these instances the actual number of organisms seemed to be very much reduced as compared with those in which a well formed necrotic center was produced.

The ganglionic layer and fiber tracts in the immediate neighborhood just beyond the outer zone of necrobiosis showed the well known signs of degeneration. The nerve cells whose fibers were involved swelled up and slowly degenerated. The neuroglia cells became much more prominent and showed mitotic figures. Their normally inconspicuous cell body increased in size, the appearance of the protoplasm became more watery and their processes, thick and rounded up. Their nuclei were often increased in number. The nerve fibers, undergoing secondary degeneration at a distance from the actual lesion, showed swelling of the axis cylinder, which broke up into granules of bizarre shape. These collected into a network with a more or less even mesh. Later they were invaded and removed by the large phagocytes. The most remarkable phenomenon about the spread of the abscess was the resistance to destruction shown by the molecular zone — quite frequently the ganglion cells beneath were destroyed completely for a considerable extent and it was possible to identify this thin strip of nervous tissue embedded in a mass of leukocytes and bacteria.

### SPREAD OF INFECTION INTO MENINGES

The meninges in the neighborhood of the lesion were quickly infiltrated with leukocytes which formed a fair degree of protection against a rapid spread of the infection in the subarachnoid space.

Where a decompression allowed the brain to herniate, compression of this space apparently favored the effectual shutting out of a generalized spread, but this factor was never demonstrated. A common complication was the separation of dura and arachnoid by a rapid accumulation of pus and bacteria. The center of such an abscess soon showed signs of necrosis and grew by addition of leukocytes to the periphery. The degree of resistance toward spread into the subarachnoid space was very high, and it was a common occurrence to find countless bacteria growing on the outer surface of the arachnoid membrane without being able to demonstrate a single organism in the subarachnoid space beneath. At such times the latter cavity was filled with leukocytes and the perivascular spaces emptying into this area were crowded with white blood cells.

When the subarachnoid space became involved it usually was filled with a coagulated exudate. It often reached a considerable distention as shown in Figure 2. Bacteria, leukocytes, and macrophages were distributed through the meshes of the pia-arachnoid. After the abscess had existed for several days, large cells with metachromatic granules, eosinophils, and plasma cells appeared on the scene. At this time the molecular zone was constantly (less often the deeper zones of the cortex) infiltrated with leukocytes, and the neuroglia cells gave evidence of proliferation. The bacteria, however, were sharply limited to the subarachnoid space, and only exceptionally began to break down the resistance of the pia mater and the molecular zone of the cortex, and to spread within the substance of the brain. It is interesting to note that only infrequently did the organisms grow into the perivascular spaces and invade the nervous tissue in this way. To repeat, the distribution of bacteria was confined by natural boundaries in the two limits of the subarachnoid space, that is, the pia mater and the arachnoid membrane. Infections spread through these thin connective tissue membranes only with great difficulty.

There was evident no natural barrier protecting the ventricles from invasion. As the growing abscess neared the ventricular wall, the ependymal lining of the latter was raised up by an exudate of serum and leukocytes. The character of the ventricular fluid was then so modified that it could be stained in section by eosin and toluidin blue. The fluid in the ventricle, which coagulated following fixation, showed no fibrin deposition. It contained a very few cells, mainly leukocytes of the mononuclear variety. The number of intraventricular cells increased with the steady progress of the bacteria toward the ventricle while the proportion of the polymorphonuclear leukocytes became

augmented. The effort of the exudative cells to localize the process to the invaded portion of the ventricle was usually partly successful, and for a time only the ipsilateral ventricle contained a cellular exudate, but the rest of the ventricle was slightly dilated with a homogeneously stained fluid. As a rule, however, the infection spread throughout the ventricular system when once the bacteria gained access; the whole system became filled with organisms and pus cells. The spread through the foramina of Luschka and Magendie resulted in a basilar meningitis which extended rapidly down the cord and into the adjacent cisterns. During this invasion of the ventricle, the choroid plexus maintained a fairly normal appearance, although large areas of the parietal ependyma and subependymal tissue became excoriated. A few polymorphonuclear leukocytes infiltrated the connective tissue stroma of the choroid and the vascular plexus was slightly dilated; but in spite of the fact that the ependyma covering the choroid plexus was bathed in pus and bacteria, but little degeneration occurred. After a time the stroma became filled with a massive cellular exudate so that the plexus was greatly thickened. A few bacteria were found within the epithelial covering and the latter began to swell up and separate from the stroma. Shreds of detached epithelium could then be seen undergoing degeneration in the debris filling the ventricle. More often the death of the animal ensued before total destruction of the choroid plexus took place. Where the original wound communicated with the ventricle the entire ventricular system became quickly dilated with a compact cellular exudate and the sero-fibrinous character of the exudate was much less marked.

### REPAIR

There were no experiments in this series which might be regarded as intermediate steps leading to the elimination of the process. Thirteen of the thirty-five animals died within forty-eight hours; sixteen died within a week but lived more than forty-eight hours, and three died nine days after the injury. There were only three that survived; these animals were killed at the end of two, three and four months, respectively. In the first instance, the cerebral lesion was irrigated with chloramin solution on several occasions, and the gross picture was that of a solid replacement of the destroyed nervous tissue by connective tissue without the usual formation of small cysts. In this specimen there were streaks of brilliant salmon pink radiating from the meninges down into the substance of the brain. The brains of the other two animals which survived showed cysts of various sizes on cross-section.

The original volume of the brain was much diminished where the wound occurred and the lateral ventricle showed a dilatation at this point (Fig. 4). In all three of these specimens the leukocytes had disappeared from the scene entirely. Strong bands of fibrous tissue arising from the fused dura and pia-arachnoid radiated into the area of destruction, carrying with them vessels from the meningeal system. Between this connective tissue replacement and the normal nervous tissue was a zone of gliosis. The phagocytic cells were still present in considerable numbers, being heavily laden with pigment. No glial cells could be found within the scar-tissue itself. The cysts were outlined by connective tissue and were often filled with macrophages. Often these cavities were in direct communication with the perivascular space. In one of the animals, the scar tissue replacement showed that the lateral ventricle had been penetrated, but the break in continuity of the ventricular boundary had been subsequently sealed over completely. None of the cystic dilatations around the perivascular spaces communicated with the ventricles. The ependymal lining at the end of four months had not yet begun to cover up the defect caused at the time of injury. The pattern of the subarachnoid tissue spaces was carried into the defect from the outside (Fig. 4), and resulted in a considerable widening out of the subarachnoid space at this point. None of these cystic enlargements seemed to communicate with the subarachnoid space. Every one of the animals which survived the infection showed a healed process in every way comparable to the healing of a sterile ablation of a similar amount of brain tissue. In none of them was found a walled-off process similar to the chronic abscesses encountered in extensions from the middle-ear or the solitary encapsulated abscesses in human beings.

## DISCUSSION

The problems confronting us in acute traumatic abscesses of the brain are primarily two: (1) the factors influencing the development of a destructive bacterial invasion of the brain, and (2) a repair of the lesion. The first comes under the head of inflammation, while the latter has to do with the re-establishment of a viable organism. Concerning the repair of brain lesions, considerable experimental work has been carried on, and the histology of the events leading to the establishment of a physiologic equilibrium is fairly well understood. A complete restoration of destroyed nervous tissue is never attained, and even in new-born animals the nervous system is too completely differentiated to reproduce any new elements. Function can be par-

tially taken over by other cells, and when confined to certain areas of the cerebral cortex a normal individual, to all intents and purposes, may be preserved. The final repair is almost exclusively a partial replacement by connective tissue accompanied by a new ingrowth of vessels and to a very slight extent by the neuroglia which interposes a zone of sclerosis between the fibrous scar and the normal nervous tissue. An understanding of the factors involved in the destruction of the nervous elements, along with the efforts to halt its spread and the removal of the debris, namely, inflammation, will give us a clue to a rational therapy.

Experimentation which would show only the processes concerned in inflammation to the exclusion of those concerned in repair is manifestly impossible. Every experiment in inflammation must, of necessity, include a varying mixture of both these phenomena in the same specimen. On the one hand must be considered the bacterial agent elaborating toxins which diffuse into the tissues; the effect is not limited to the area directly reached by the poison but extends for a great distance by secondary degeneration of the affected neurons. On the other hand, there are the countless defensive cells poured out from the vessels in an effort to halt the spread of the destructive agents; the removal of dead material by the cells of the macrophage class; and finally, the reparative efforts of the young fibroblasts. All of these processes are intermingled and exist in varying proportions in different localities of the wound. In addition to these generalizations, there must be taken into account the factors peculiar to the central nervous system — absence of fascial planes, the effect of the chemical constituents of the degenerating tissue, the ease with which the brain can distend, and the effect of the rigid envelop which permits of little expansion — in a word, the anatomico-physical characteristics of the affected organ.

The division of the pathologic process into three zones serves a useful purpose in presenting the microscopic description in a more or less orderly way. As an active process, another kind of division would perhaps furnish us with a more logical conception of a traumatic brain abscess. This would consider but two zones: (1) a central necrotic foreign body, the source of soluble toxins, and (2) a peripheral zone in which the struggle between two opposing forces takes place. In this latter zone, which is a combination of the zone of cellular exudate and necrobiotic zone, the course of the abscess is determined: the termination may be either (1) a losing fight in which the animal as an organism succumbs, or (2) a retardation of the spread until a connec-

tive tissue capsule may be formed, or (3) an actual neutralization and elimination of the bacterial agent. In these infections where the streptococcus was present, the central core became in every sense a foreign body. Encircling this the organisms grew luxuriantly and formed the reserve depot from which a spread forward could be made in case the tissues were sufficiently weakened. The outer zone represents the vascular changes and associated exudation met with in inflammation. The diffusion extends from the central mass into the periphery, with a progressively weaker effect as it diffuses from the point of elaboration. The structural tissue is killed or damaged and the blood vessels become unable to hold within their walls the fluid and nonameboid elements. This zone of exudate and hemorrhage becomes the arena in which the fate of the animal is determined. If the virulence of the organism is lower than the combined resistance of cellular exudation and development of antitoxic bodies, then the so-called zone of "red softening" turns progressively to one of a salmon yellow, and finally, to one of brown color. If the relative strengths of the infection and resistance are reversed, the bacteria grow forward into the outer zone which is progressively incorporated into the necrotic center. In some types of infection the tendency of the red cells to spill out into the tissues is not marked. Just how this factor modifies the spread of the infection will be discussed later. The ecchymosis of red corpuscles is in all probability a weak chemical protection and ought not to be considered as a part of a preparation for the formation of a purulent inflammation. The notion that an abscess is always preceded by "red softening" gives a false impression in that it makes the effect appear to be the cause. In no wise does it help our conception of the processes going on in the development of a purulent focus. The evidence points away from the idea that red softening is in any way a "preparation" of the nervous tissue for the development of an abcess.

The kind of infection used in these experiments may be considered under two heads: (1) pure cultures, and (2) pus. The former always proved to be the weaker in virulence, while the latter probably owed its destructiveness to the associated agressins. By passing the pus from the cortex of one animal to another, a rapidly fatal lesion could be produced, with death within twenty-four hours. In these instances the effectiveness of the toxin was shown in the widespread "burnt-out" appearance of the tissue as well as the uninterrupted advance of the organisms. Their distribution was more or less uniform throughout the abscess and extended for a short distance beyond the cellular

exudate into the zone of necrobiosis. When the virulence was not so great, the bacterial invasion was sharply confined to the central zone of necrosis. The failure to spread invariably into a purulent meningitis from the point of injury is interesting. Rarely does the subarachnoid space contain organisms at any considerable distance beyond the lesion. On the other hand, the subarachnoid space or brain tissue may not be involved even when the subdural accumulation of pus reaches enormous proportions. The occurrence of a lepto-meningitis follows much more often a spread from the ventricles through the foramina in the hind-brain. This spread into, or direct inoculation of, the ventricle is one of the most serious complications of a brain abscess.

The natural resistance to the spread of infection is very inadequate. The only barriers at all comparable to the fascial planes elsewhere, are the meninges. The essential elements taking part in this protection are the cells making up the pia-arachnoid. Their behavior toward debris introduced into the subarachnoid space has demonstrated the active rôle played by these mesothelial cells. Under the stimulus of products of tissue destruction, they proliferate and give rise to ameboid mononuclear cells with marked phagocytic powers (Essick[14]). Figure 17 shows them massed at the glued edges of the meninges bordering a brain abscess. By removing the destroyed tissues they help to keep the meningeal spaces open so that a circulation of cerebro-spinal fluid is possible. The abscesses following extension from the air sinuses (middle ear, frontal, etc.), meet with a definite obstruction in the dura, but the more cellular leptomeninges are extremely resistant to a spread at right angles to their plane. The delicate arrangement of the closely interweaving fibrils in the pia mater and the arachnoid membranes keeps the process on either side of them very sharply localized. Thus an encephalitis is halted at the pia and prevented from becoming an uninterrupted meningitis; a subdural abscess is similarly checked by the arachnoid membrane. The massive growth of bacteria beneath the dura may not only cover a large part of one hemisphere, but also extend beyond the mid-line beneath the falx-cerebri, and yet if one searches carefully it may be impossible to find a single organism within the cellular exudate in the subarachnoid space, whereas the outer sur-

14. Essick, C. R.: Formation of Macrophages by the Cells Lining the Subarachnoid Cavity in Response to the Stimulation of Particulate Matter, Carnegie Institution of Washington, Publication 272, 1919. Contributions to Embryology, **9.**

face of the arachnoid membrane is literally covered with them. To be sure, this resistance may be broken down, but only after the tissues have been grossly insulted. The normal amount of connective tissue within the substance of the brain is almost negligible, being confined to the thin sheaths of the vessels.

The rich, close-meshed capillary plexus within the gray matter may be one factor in determining the relatively greater resistance of the gray matter to infection as compared with the white matter. The latter is provided with a system of elongated capillary loops so that the blood supply is very much poorer than in the cellular layers. This disparity in resistance of gray and white matter is further heightened by the fact that the central end of an injured neuron may live, but the peripheral end always dies. The nuclei of the cells making up the fiber tracts are situated largely in the cortical gray and the secondary degeneration in the peripheral end of the nerve fiber furnishes a good culture medium for the bacteria.

The most efficient protection for the substance of the brain is that supplied by the leukocytes which pour out into the perivascular spaces around the injury and quickly form a cellular barrier against the spread of the infection. Probably the injury to the tissues is the primary stimulus in the case of the ordinary infection, but shortly after an absorption of the toxic substances makes increased demands on the white blood elements. Long before any antibodies may be formed, the bacteria have created quite an area of necrosis at the point of injury. The blood vessels become dilated and enormous numbers of leukocytes crowd the perivascular spaces. White blood cells emigrate from all the small vessels in the neighborhood and crawl toward the lesion, preferably along the perivascular spaces. If the infection be entirely meningeal, the perivasculars are transformed into veritable crowded highways by the white cells hurrying toward the subarachnoid space. Such "perivascular exudate" should not be viewed as an invariable sign of encephalitis, but rather as the natural pathway for inwandering cells. These perivascular spaces would make excellent natural channels for the spread of organisms through the nervous tissues were it not for the fact that the advance of the bacteria is very early blocked by the cells coming into these perivascular channels from the blood-stream. Occasionally one comes on a vessel that has become thrombosed, and then it furnishes an excellent pathway for the spread of the bacteria. Inasmuch as the leukocytes in such a case do not continue to come out of the vessels into the perivascular spaces, the latter become literally filled with colonies of bacteria (Fig. 9). The leuko-

cytes, then, both of the polymorphonuclear and the small mononuclear variety, are the best protection that the brain may acquire quickly. The predominating type of cell cannot be predicted, and the variety of the cell seems to be entirely independent of factors under control.

There is unquestionably some development of antibodies in the blood stream, but in this type of abscess, protection from such a source must be a small factor in overcoming the process. A far better chemical protection is to be found in the escaping whole blood which occurs in the necrobiotic zone in considerable quantities. The efficacy of this mechanism has been demonstrated in several ways in this series of experiments. In one animal in which a button of bone was pushed into the wound it was seen at necropsy that a very large hemorrhage had developed (Fig. 16). Microscopically, this portion of the injury proved to be absolutely unaffected by the infectious process, although this had involved a considerable amount of the cerebral hemisphere. Not only does such an area of hemorrhage remain uninvaded by the organisms in the midst of so much infection, but also the extravasated corpuscles are actually being removed by the phagocytes while young fibroblasts and blood vessels sprouts are beginning to repair the defect. Another remarkable instance of the protective powers of blood is shown in Experiment 66. Owing to the scarcity of animals when experimental abscesses were made, a good many of the cats had to be used for more than one observation. After each experimental procedure they were allowed to live for several weeks and to regain their "normal" appearance. After serving as the subject of three experiments on the central nervous system and recovering, this cat, while under ether, was struck on the head with a sharpened nail and the wound infected. Gross necropsy showed that the puncture had been made so close to the mid-line that a large meningeal vessel had been opened up and a hemorrhage had covered the left cerebral cortex for at least 2 cm. from the brain wound. There was no evidence anywhere of a meningitis macroscopically. The animal had lived for three days, and when studied microscopically showed an extensive destruction of brain tissue. The bacteria were growing in enormous numbers along the natural pathways in the nervous tissue. There was no cellular zone visible under low magnification, and with higher powers, a few inwandering polymorphonuclear leukocytes were observed as single cells, scattered around the edges of the abscess. Apparently, the previous experimentation had in some way broken

down the responsive cellular exudate and the bacteria proliferated within the brain much as if they had been planted on cultural media. In this instance the lesion was absolutely localized to the substance of the brain, and not a single organism could be found growing within the subarachnoid space. The whole blood formed a perfect barrier against a spread of organisms in the meningeal spaces and constituted the sole protection against a meningitis.

Most of the experimental abscesses were marked by a varying tendency to ecchymosis. The actual number of red blood corpuscles was usually not great. Such small accumulations of erythrocytes offer a momentary obstruction to an uninterrupted advance of the infection. The more susceptible areas allow the infection to work around the sides of the blood extravasation. Thus in Figure 7 the perivascular hemorrhage stands out as a salient, retarding the line of advancing bacteria. Finally, such accumulations of red corpuscles are completely surrounded and gradually are included in the necrotic core. This may help us to understand the value of the treatment of penetrating wounds of the brain at the battle front. The best results have followed extensive lavage of the tract and tight closure of the wound. The chemical value of hemorrhage seems to lie in the red blood corpuscle rather than in the serous or the fibrinous exudate.

Of certain value is the relief of pressure developing in a confined space. Increase of brain bulk is unquestionably the beginning of a vicious circle and is the worst complication which follows a traumatic infection. The method of combating the rising pressure within the brain by opening the skull and dura has been employed by surgeons for a long time. Certainly, it prevents the dislocations if enough outlet is given to the expanding brain, as is seen in Figure 19. The most distressing feature follows the trephining of the skull and the formation of a sloughing and infected fungus. It is to be hoped that the reduction of brain-bulk following the alteration of the tonicity of the blood will give a practical method of removing the cause, instead of trying to combat the effect. A very suggestive method of modifying the development of edema and its correlated increase in intracranial pressure may be found in the work of Weed and McKibben.[15] It may be that the effects of trauma can be overcome by suitable intravenous injections of hypertonic solutions of salt or dextrose, and the phe-

15. Weed and McKibben: Experimental Alteration of Brain Bulk, Am. J. Physiol. **48**:531 (May) 1919.

nomena of swelling be temporarily, at least, if not permanently obviated.

The factor of injury in traumatic abscess of the brain is most important from two standpoints. The most obvious is the destruction of brain-continuity and its associated secondary degeneration. Less obvious, but more important, is the swelling of the injured portion of the brain following such injuries. The most careful manipulation of the normal brain is sufficient to set in motion the factors increasing the size of the organ. These causes are little understood, except for the general concept of disturbance of balance of the tissue fluids, the intercellular, cerebrospinal and intracellular fluid. Edema of the brain, when the skull and dura are unopened, causes a rise in pressure of the contents of the cranium and must facilitate the thrombosis of vessels in the injured area. The actual blood supply is seriously interfered with at a time when there should be a protective hyperemia. The resultant dislocation of so delicate a mechanism caused by the swelling of one region and a compression of the rest is a most serious complication. Added to this phenomenon is a ventricular dilatation which further reduces the available space for normal tissue. Such a complex results in an increased susceptibility to destruction where a bacterial agent is present, and serves to prolong the edematous condition initiated with the original wound. The relief of such a condition by trephine merely transforms one complication into another which is little less of an evil. The infected hernia seems to have no end to the possibility of expansion. It cannot be too strongly emphasized that this phenomenon of injury, and its associated swelling, is the essential difference between traumatic brain abscesses which run such an acutely destructive course and the slow-growing abscesses which extend from the air passages. Operative procedures aimed at the cure of the slowly growing abscesses must scrupulously avoid all unnecessary disturbance of the healthy brain tissue, lest they excite the phenomena of brain swelling and transform the chronic abscess into an acute traumatic one.

The products of secondary degeneration of the fiber tracts offer an excellent medium for the culture of bacteria. The presence of the fatty derivatives of myelin must have a decided influence on the growth of the organisms as well as retard the interchange of fluids between the necrotic center and the surrounding tissues. These experiments with foreign bodies were too few to draw any conclusions as to their

effect on the progress of an abscess. Infection of the ventricle seems to bear no relation to the general reaction of the animal, whether inoculated directly or becoming involved by a secondary spread. In the thirty-five experiments recorded here, the length of life seemed to be governed by the virulence of the infection. One of the cats surviving the infection showed that the original wound had penetrated the ventricles and reached the surface of the cortex on the side of the brain farthest away from the entrance of the perforator.

As far as can be determined the nervous tissue has practically no natural planes in which infection may spread with greater difficulty. The neuroglia forms a feltwork which makes the gray and white matter practically homogeneous. Secondary degenerations furnish a decrease in resistance, but this cannot be looked on as preformed. The perivascular spaces are natural channels which can be invaded by bacteria. Unless the normal protective mechanism is disturbed, either by interfering with the response of the white blood cells, or by thrombosis, such channels cannot be entered by the bacteria. The spread of an infection occurs by direct destruction of the tissue assisted by the secondary degenerative processes formed by nerve fibers separated from their cells of origin.

Mingled with the processes of destruction and protection are cells whose main function is to remove the debris. The large phagocytic cells appearing in such enormous numbers in the central nervous system were regarded by the early writers as pathognomonic of inflammation. In no inflammatory process elsewhere in the body do they appear in like numbers. The formation of such large phagocytic elements from the cells of the arachnoid, from those of the vascular adventitia and from neuroglia cells has been demonstrated to occur under the influence of altered environment. These transformed cells gather in the region just beyond the zone of leukocytes as long as the infection is active, greedily ingesting the free red cells, dead leukocytes, and products of destruction of the nervous tissue. If the organisms are killed and their toxins neutralized, these macrophages invade the necrotic core and remove it. Long after the last leukocyte has left the scene these large cells are found in great numbers, with their bodies distended with ingested material. The removal of the debris is exceedingly slow in the brain, since it depends entirely on the ameboid activity of these large mononuclears. The lack of a lymphatic system, which, in the infections of the tissues of the body generally, plays an important rôle in the removal of waste products of inflamma-

tion, may easily account for the increased demand for these cells in processes involving destruction of the nervous system. Where the abscess spreads rapidly, these phagocytes appearing beyond the leukocytic wall are destroyed, and as a result, few are demonstrable in this type of lesion. Where the extension is slow, or where the rate varies considerably in different localities of the same abscess, these cells are often found in such numbers that they almost replace the normal structures.

The attempt to repair the lesion is instituted within the first few days. Such repair is entirely brought about by the fibroblasts and new vascular sprouts. They are the derivatives of the meningeal mesothelium and of the adventitial cells of the blood vessels. In the slowly growing abscess these efforts are partially successful and a capsule is formed and serves to retard the spread of the process to a marked degree. This mechanism is too slow to be of any assistance in stopping a traumatic abscess. These fibrous elements form, however, the tissue which replaces the lost structures, if the infection is overcome. This fibrosis of the defect is, however, frequently complicated by the formation of cysts in the scar. These develop as dilatations of the perivascular spaces and are to be explained as accumulations of intramedullary cerebrospinal fluid, the free drainage of which has been interfered with (Pick[16]). The superficial defect of the brain is partially filled by an increased development of the subarachnoid tissue, the meshes of which are in free communication with the rest of the cerebrospinal fluid spaces.

SUMMARY

Experimental abscesses produced in the cortex of thirty-five cats by injury and infection of the brain, resulted in a rapidly fatal process which simulated the traumatic injuries of the central nervous system in man. The affected part enlarged and brought about a marked dislocation and compression of the rest of the nervous tissues. In 50 per cent. of the cases the infection reached the ventricular system within a few days, and from there usually spread into a basilar meningitis through the metapores of the fourth ventricle. The tendency of the infective process to invade the subarachnoid space from the point of injury was not marked, but in a third of the animals the infection entered the subdural space, forming there a subdural abscess.

16. Pick, A.: Ueber cystöse Degeneration des Gehirns, Arch. f. Psychiat. u. Nervenh. **21**:910, 1890.

These lesions were very different from the more slowly growing abscesses extending from the air sinuses. The latter, occurring frequently in man, may be differentiated by the relatively slight swelling and dislocation, and by the development of a definite connective tissue capsule between the lesion and the sound parenchyma. The traumatic abscess in the experimental animal extended rapidly along the fiber-tracts. No encapsulation could be demonstrated in any of these observations. Healing took place by the ingrowth of connective tissue.

Plate 1—Explanation of Figures.

Fig. 1.—Photomicrograph. Abscess of three days' duration, confined almost exclusively to the fiber tracts. The center was occupied by a large bubble of gas. An extensive subdural abscess is complicating the brain lesion. (Hem.-eosin, × 3.7.)

Fig. 2.—Photomicrograph through the leptomeninges immediately adjacent to the abscess. An early stage of meningitis accompanied by an extensive serofibrinous exudate. The perivascular spaces of vessels penetrating the cortex are crowded with leukocytes. (Hem.-eosin, × 72.)

Fig. 3.—Photomicrograph of the brain tissue just beyond the leukocytic zone (top of figure). The perivascular spaces are engorged with leukocytes, practically obliterating the lumen of the vessels. (Hem.-eosin, × 77.)

Fig. 4.—Photomicrograph—healed abscess. Dura adherent to defect in cortex. Small cysts are located in the connective-tissue replacement. Lateral ventricle dilated under the injury. (Hem.-eosin, × 3.)

Fig. 5.—Retouched photograph of head of cat in which cerebral hemispheres are partially exposed; the dura is intact. The injury appears in the dark area of the left hemisphere. The brain markings are obliterated on the affected side by a thick purulent subdural exudate. This is sharply limited, medially, by the falx cerebri. The right hemisphere shows engorgement of blood vessels with a very slight loss of distinctness of brain markings. (× 1.)

Fig. 6.—Photomicrograph. Left side of picture—zone of leukocytes. Middle shows a layer of extravasated erythrocytes interposed between the lesion and the normal tissue (on the right). (Hem.-eosin, × 60.)

PLATE 1

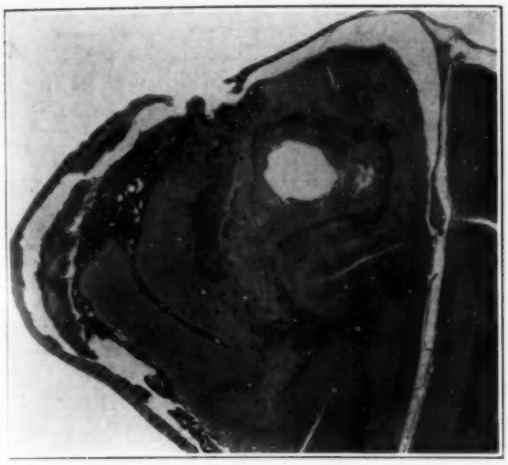

Fig. 1.

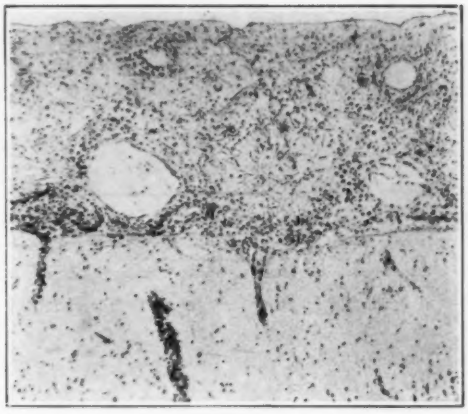

Fig. 2.

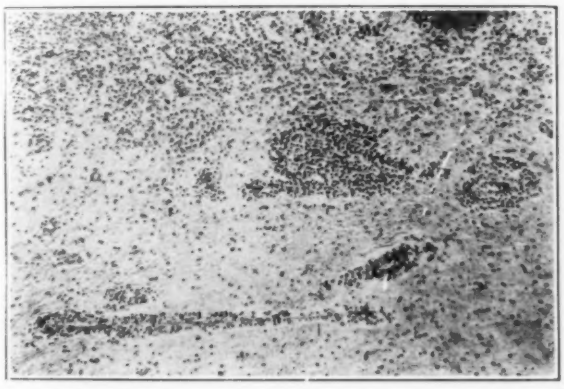

Fig. 3.

Fig. 4.

Fig. 5.

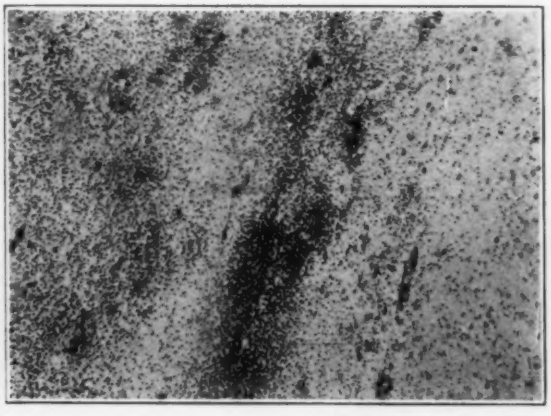

Fig. 6.

Plate 2—Explanation of Figures

Fig. 7.—Photomicrograph. The center of the figure shows two vessels, the perivascular spaces of which are filled with red blood corpuscles. The infection is momentarily halted at this point but the organisms are out-flanking them on both sides; separated from the normal tissue (right side of figure), however, by a wall of leukocytes. (Hem.-eosin, × 60.)

Fig. 8.—Compression and dislocation of brain by a large subdural abscess. The left lateral ventricle is slightly compressed and the right correspondingly enlarged. The white fiber tracts on the left side show the early development of a translucent gray core. (Drawing × 1.4.)

Fig. 9.—Photomicrograph of abscess showing the rapid extension of the bacteria when unimpeded. All the darker masses represent conglomerate colonies of organisms — completely filling the perivascular spaces. (Hem.-eosin, × 60.)

Fig. 10. — Photograph of sections through a brain in which a rapidly developing abscess of two days' duration has spread into the ventricle. The left lateral ventricle is distended with inspissated pus while the right is dilated with a clear coagulum. This shows a remarkable dislocation of the left hemisphere and a compression of the right. (× 1.2.)

PLATE 2

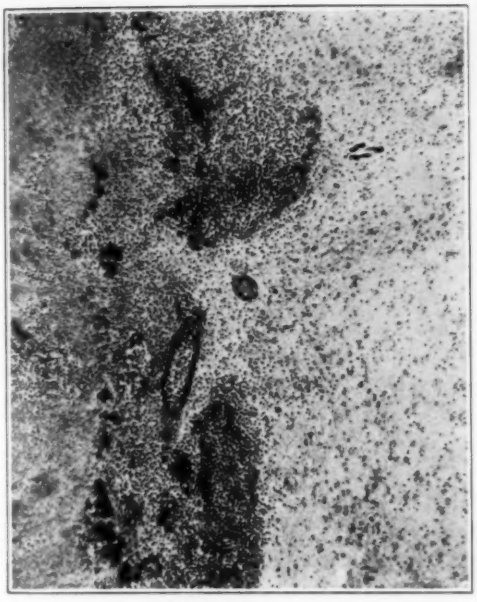

Fig. 7.

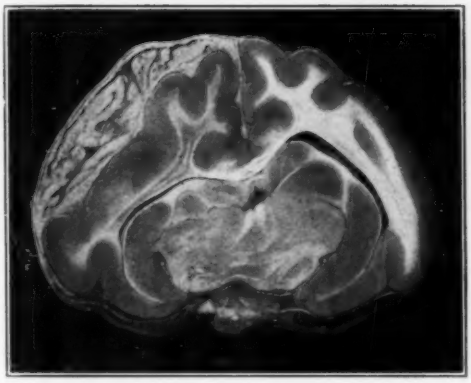

Fig. 8.

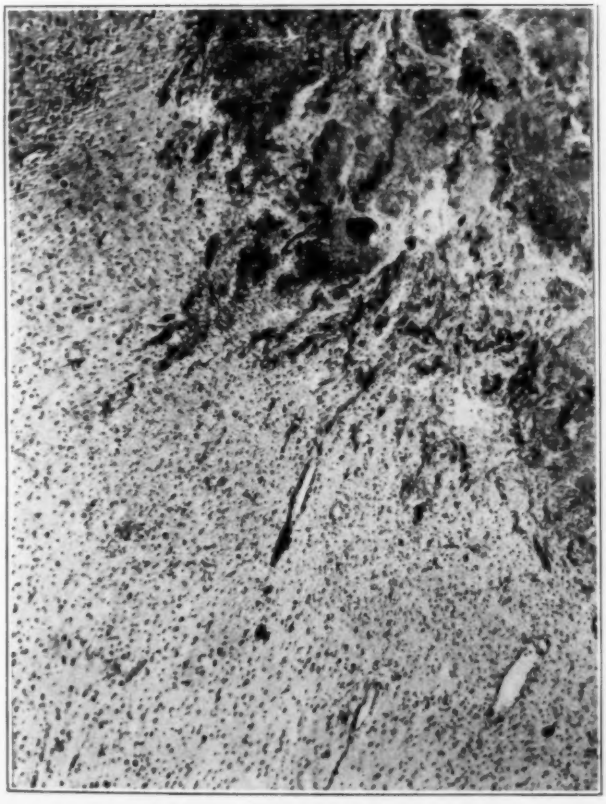

Fig. 9.

Fig. 10.

Plate 3—Explanation of Figures

Fig. 11.—Photograph. Sections through cat's brain showing the invasion of the fiber tracts by an abscess. This lesion is accompanied by a blood-tinged exudate and photographs black. (× 1.2.)

Fig. 12.—Cyst forming within an abscess. The entire white matter is translucent on the affected side so that the distinction between cortex and fibers is almost obliterated. (Drawing × 1.2.)

Fig. 13.—Photomicrograph. The spread in extremely rapid as shown by the thickness of the zone of leukocytes. In this specimen bacteria actually are found beyond the sharply delimited wall of leukocytes. Death of animal occurred in three days. (Hem.-eosin, × 60.)

Fig. 14.—Photomicrograph showing the lamination of a slowly developing abscess and the predilection for the white fiber tracts. The three indentations in the periphery of this process are the seat of perivascular hemorrhages. The large subdural abscess has separated from the leptomeninges. (× 3.9.)

Fig. 15.—Photomicrograph showing the grouping area of organisms and their mode of breaking through the leukocytic defense. The upper part of figure is the necrotic core. Extending in two broad columns are the two waves of bacteria; the one on the left is separated from the necrobiotic zone (bottom of figure) by a very few leukocytes. (Hem.-eosin, × 60.)

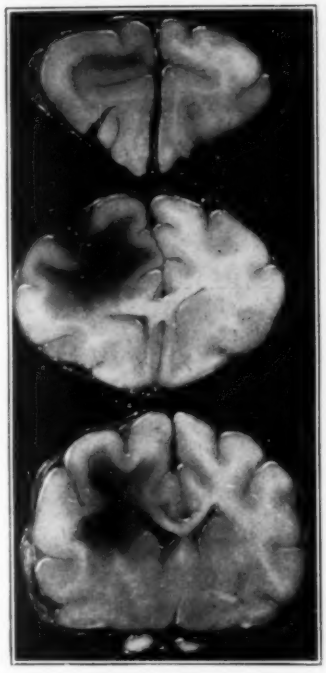

Fig. 11.

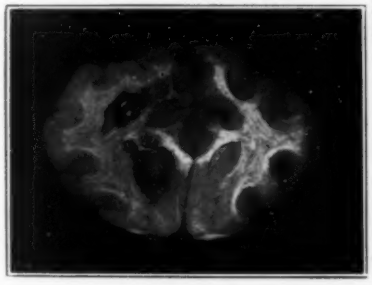

Fig. 12.

Fig. 14.

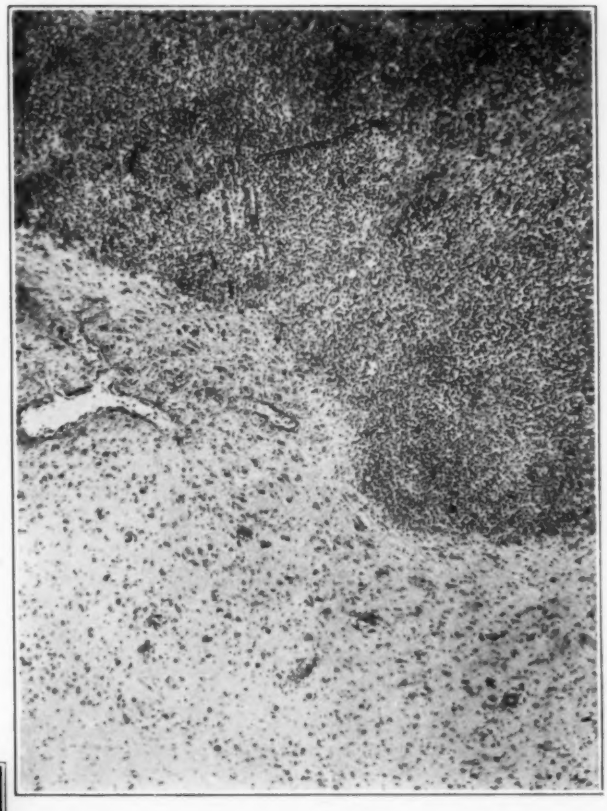

Fig. 13.

Fig. 15.

Plate 4—Explanation of Figures

Fig. 16.—Photomicrograph of a transverse section through entire cortex of cat. The abscess has developed within the white fiber tracts and into the thalamus. An extensive hemorrhage at the base made an efficient barrier to the spread in this direction. A large part of the blood has already been removed. This process lasted nine days. (× 3.3.)

Fig. 17.—Photomicrograph of a wall of macrophages derived from the pia-arachnoid which is shown blocking the subarachnoid space and preventing the destructive infection (upper part of figure) from spreading into a meningitis. (Hem.-eosin, × 60.)

Fig. 18.—Photograph of section of cat's brain in which the original wound communicated with the ventricular system. The lateral ventricles are dilated with pus. (× 1.4.)

Fig. 19.—Section through abscess developing under a trephine hole made in the skull. Remarkable hernia of left side of brain. The displacement is very great on account of a rapidly developing subdural abscess. Right hemisphere not compressed, as shown by midline. Six days' duration. (× 1.3.)

Fig. 16.

Fig. 17.

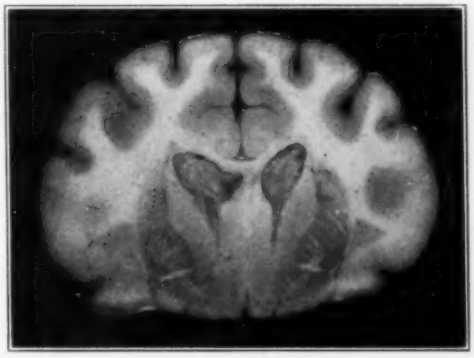

Fig. 18.

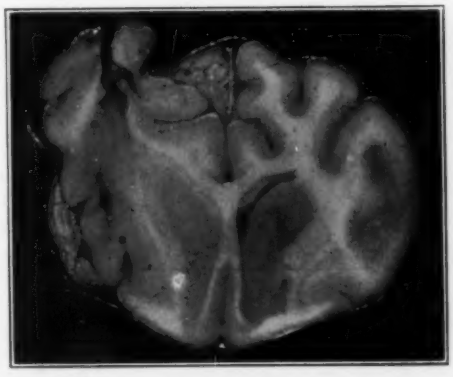

Fig. 19.

# HISTOPATHOLOGY OF CARCINOMA OF THE CEREBRAL MENINGES *

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CHICAGO

It is generally admitted that carcinoma of the nervous system is always a secondary process, spreading either from neighboring affected regions, or reaching the brain and its meninges by way of metastases. There have been recorded a few cases of so-called primary carcinoma of the brain (Gedge-Latham,[1] Rustizky,[2] Russel,[3] and Coats[4]), but these cases proved to be, on closer examination, some other varieties of tumors, like glioma, endothelioma or sarcoma.

The statistics by Buchholz,[5] Gallawardin and Varay,[6] Kaufman,[7] Krasting,[8] Fischer and De Foy[9] not only demonstrate the relative frequency of metastatic growth of cancer in the brain, but also that it may originate from any organ in the body, no matter how remote. Thus, it may come even from the prostate, rectum or ovaries, and make its first appearance long after the original growth has been removed. In Guttman's[10] case the brain showed metastases eighteen years after an operation for ovarian carcinoma, and in Gallawardin's case,[6] eight years after the primary tumor was located (in the subclavicular space). In this case about 200 nodules were found in the substance of the brain and spinal cord. In the majority of recorded cases distinct gross or microscopic nodules were found which could very well account for the various clinical symptoms, as hemiplegia, monoplegia, aphasia, jacksonian epilepsy, single or combined nerve paralyses, various psychical disorders, etc. But cases have been reported in which no metastases in the brain tissue could be demon-

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* From the Laboratory of Pathology, Cook County Hospital.

1. Gedge-Latham: Primary Carcinoma of Brain, Brit. M. J. **1**:284 (March 27) 1869.
2. Rustizky: Virchows Arch. f. path. Anat. **59**:191, 1874.
3. Russel, J.: Brit. M. J. **2**:709 (Dec. 2) 1876.
4. Coats, J.: Primary Carcinoma of Brain, Brit. M. J. **1**:959 (May 4) 1888.
5. Buchholz: Monatsch. f. Psychiat. u. Neurol. **4**:183, 1898.
6. Gallawardin and Varay: Rev. de méd. **23**:441, 1903.
7. Kaufman, E.: Cor.-Bl. f. Schweiz. Aerzte **36**: 1906.
8. Krasting, Karl: Same as Footnote 7. Published in extenso in Ztschr. f. Krebsforsch. **4**:315, 1906.
9. Fischer and De Foy: Ztschr. f. Krebsforsch., 1905, p. 195.
10. Guttman, C.: Fortschr. d. Med., 1904, No. 4, p. 141.

strated, in spite of the presence of marked clinical symptoms of brain lesion. Such cases have been explained by Oppenheim[11] as toxemic, that is, caused by the cancer toxins, just as uremia or diabetes may produce clinical symptoms of brain lesion without any demonstrable anatomic changes.

Oppenheim's view was also shared by Nonne[12] and Siefert.[13] However, cases of supposed cancerous toxemia became practically unknown after thorough microscopic studies were made not only of the brain substance, but also of the meninges. It was found that the brain tissue itself may be totally free from cancer elements, while the dura or pia-arachnoid is so infiltrated as to cause serious symptoms such as hemiplegia, aphasia, etc. The infiltration may be of the dura only, causing it to appear thickened and hemorrhagic, resembling so-called hemorrhagic internal pachymeningitis. In fact, such cases have been described as "pachymeningitis carcinomatosa" (Westenhöfer,[14] Dahmen,[15] Lissauer[16]). In other cases there was no real involvement of the dura, but rather of the pia-arachnoid. The latter group of cases have been described as "meningitis carcinomatosa" (Siefert,[13] Saxer,[17] Scholz,[18] Stadelman,[19] Krause,[20] Schwarz and Bartels[21]). These two terms, pachymeningitis and meningitis, imply an inflammation of the corresponding cerebral membranes, and some authors[14-b] claim that there is actually present an inflammation caused by the presence of cancer cells.

Another point deserving attention is how cancer cells reach the brain or its membranes. Kaufman[7] pointed out that when the metastasis occurs by way of the blood stream, the brain tissue is principally

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11. Oppenheim, H.: Ueber Hirnsymptome bei Carcinomatose ohne nachweisbare Veränderungen im Gehirn, Charité-Ann. **13**:335, 1888.

12. Nonne, M.: Discussion of Saenger's Cases in Neurol. Centralbl., 1900, p. 187.

13. Siefert, E.: München. med. Wchnschr., May 20, 1902, p. 826.

14. Westenhöfer: (a) Pachymeningitis carcinomatosa hemorrhagica interna productiva, Virchows Arch. f. path. Anat. **175**: 1904. (b) Deutsch. med. Wchnschr., 1903, No. 46; Soc. Proc., p. 359.

15. Dahmen, Z.: Pachymeningitis carcinomatosa, Ztschr. f. Krebsforsch. **3**:300, 1905.

16. Lissauer, Max: Zur Kenntniss der Meningitis carcinomatosa, Deutsch. med. Wchnschr. **37**:16 (Jan. 5) 1911.

17. Saxer: Ueber dem Bilde einer Meningitis verlaufende Carcinomatöse Erkrankung der Gehirnhäute, Verhandl. der deutsch. path. Gesellsch. **5**:161, 1903.

18. Scholz, W.: Meningitis carcinomatosa, Wien. klin. Wchnschr., 1905, No. 47, p. 1231.

19. Stadelman, E.: Berl. klin. Wchnschr., 1908, No. 51, p. 2262.

20. Krause: Discussed in Deutsch. med. Wchnschr., 1903, No. 46, Soc. Proc., p. 359.

21. Schwarz and Bartels: Deutsch. Ztschr. f. Nervenh. **42**:85, 1911.

affected, but when it travels through the lymphatics the meninges become involved. But he does not give the precise mode of invasion of the intracranial spaces. These two problems — the reactive phenomena in the meninges to the presence of cancer cells, and the character of the meningeal involvement — I have studied in the following case.

REPORT OF CASE

*History.*—A woman, aged 40, housewife, entered Cook County Hospital on the surgical service of Dr. Cubbins, April 5, 1918, with a history of some malignancy of her left breast which had been removed a year previously. About six months later a small solitary gland which appeared in the left axilla was removed. Four months after the last operation she started to complain of severe headache which was constant and accompanied by nausea and vomiting. The vomiting had no relation to meals and was never bloody or coffee-ground in character. She also complained of numbness in the left arm and right corner of the mouth. Other diseases were denied. Her family history was negative.

*Examination.*—Examination revealed a well nourished, white woman, with surgical scars in the left chest and left axilla. Tumor masses, cervical or axillary adenopathies were not found. The skin, tendon and pupillary reflexes were normal and sensibility was unimpaired except over a small area below the right corner of the mouth, which was analgesic. Vision was 10/10 in the left, 4/10 in the right eye; the fundi were normal. The pharynx, larynx and nasal accessory sinuses were also normal. Neither were there any abnormalities of the genito-urinary organs, speech, mentality, gait, etc.

*Course.*—After a few days the patient was transferred to the medical service of Dr. Frederick Tice. There she began to complain of severe pain in the epigastrium, about two hours after meals, and slight drooping of the right eyelid set in, with convergent strabismus. The ptosis progressed, the fundi showed a beginning papilledema, the numbness spread over the right temporal region, cheeks and eyes, and the convergent strabismus became more marked. The spinal fluid was under increased pressure, of normal color, with three lymphocytes per cubic millimeter, and positive Nonne. The urine contained no albumin, but showed epithelial cells. Roentgen examination of the stomach and sella turcica was negative. Test meals showed no gastric trouble and a brain tumor of indefinite location was suspected.

*Operation.*—May 4, 1918, that is, a month after the patient entered the hospital, a decompression operation was done by Dr. Carl Meyer. The dura was found thickened and covered with nodules. One of them was removed and proved to be, on microscopic examination, a carcinoma. A particle from the amputated region of the breast also was removed and likewise proved to be carcinomatous (alveolar). Three weeks after the operation the patient died.

*Necropsy.*—The postmortem by Dr. John Nuzum revealed, among other things, primary medullary carcinoma of the left breast, secondary carcinoma of the axillary, mediastinal and anterior cervical lymph glands, carcinosis of the visceral and parietal pleura, secondary carcinoma of the liver, left suprarenal body, metastases of the meninges . . . recent surgical trephine defect in the right temporal and parietal bones, etc.

Further examination of the brain showed the right hemisphere covered by an apparently thickened dura which was loosely attached to the pia and easily removable. The thickness of the dura was about a quarter inch over the parietal region, gradually decreasing toward the frontal and occipital lobes. The outer surface of the thickened dura was somewhat rough in places because of the nodules, while the inner surface was represented by a pseudomembrane that consisted of numerous nodules of various sizes. Some of the nodules were colorless, some hemorrhagic. The pseudomembrane was solidly merged with the dura, from which it easily could be distinguished even with the naked eye, but from which it could be separated only with a very sharp knife. The vessels of the dura were much congested, distended and quite prominent. The pia on the same (right) side was very opaque, somewhat thickened, especially over the sulci, showed many distended veins, but no roughness of the surface, no hemorrhages, no nodules, no adhesions to the dura or to the adjacent portions of the brain. The cerebral sulci were not distended, the convolutions not flattened. The base of the brain, the cerebellum and the basilar nerves showed no abnormalities. In marked contrast to these findings, there was nothing abnormal in the left dura, left pia-arachnoid, and various sections through the hemispheres revealed nothing pathologic.

*Microscopic Examination.*—The microscopic examination covered various portions of the diseased dura, pia-arachnoid, brain tissue proper, the basal pia-arachnoid surrounding the left third nerve, both sixth nerves, as well as similar portions of the healthy side of the brain and its coverings. The celloidin and frozen sections were stained with toluidin blue, Van Gieson, hematoxylin-eosin, Herxheimer and Mann (Alzheimer Method V). The dura changes were studied on transverse and surface sections.

In the neighborhood of the longitudinal sinus (Fig. 1) the dura showed, on transverse sections, a practically normal structure, namely, numerous lacunae and interspaces between the connective tissue fibers. The latter showed distinct nuclei in the form of oval bodies which could easily be distinguished from other formations to be described later. The connective tissue separating the lacunae exhibited no pathologic elements, while the interspaces were invariably packed with red cells, large masses of "epithelial" cells and so-called "gitter" cells. Interspaces free from any contents were exceptionally rare, in contrast to the lacunae which, as I said, were always empty and devoid of any contents. The deeper layers of the dura and the pseudomembrane itself appeared areolar, exhibiting numerous vacuoles formed by connective tissue meshes and packed with conglomerations of cancer cells, large necrotic masses, and fibrin.

The carcinomatous cells usually formed distinct foci, were often blended with each other, forming one mass of cytoplasm containing sometimes as many as eight or ten nuclei (giant cancer cells). Some of the cells showed a broken-up cytoplasm which occasionally appeared vacuolated, with a dislocated, chromatin-rich nucleus. In many cells even the nucleus was broken up into small granules, and in some the nucleus was totally lacking. Some cells contained granules of hemoglobin and when stained with scarlet red showed large quantities of fat globules. The necrotic masses were very large, appeared homogeneous and stained badly, the cell bodies and their nuclei being very indistinct. Scarlet red stain showed in them also the presence of fat. Fibrin was present in many vacuoles, together with hemorrhagic foci, or scattered red blood cells.

The alveolar structures of the dura could be much better followed up on surface sections which excellently exhibited the alveoli and the enclosed conglomerations of the carcinomatous cells, as well as the interspaces (Fig. 2) and their pathologic contents. In such sections it was easy to see that the foci of cancer cells always lay freely within the alveoli, never touching their walls, from which they were always separated by a free space. The interspaces also showed very distinctly, filled like the alveoli with cancer cells, erythrocytes and so-called "gitter cells." The latter were round in shape, contained numerous small round vacuoles with an excentrically located, flattened nucleus. Stained with scarlet red they invariably showed the presence of a fat-like, lipoid substance.

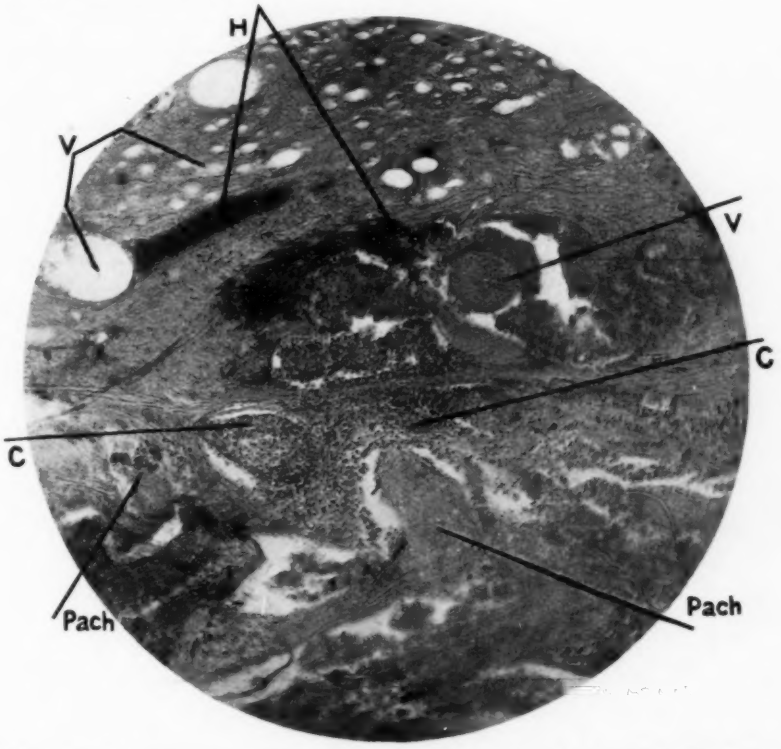


Fig. 1.—A transverse section of the dura near the longitudinal sinus. *L*, lacunal numerous cavities, covering a practically normal dura in the upper portion of the photomicrograph; *H*, hemorrhagic foci in some of the interspaces; *C*, carcinomatous foci with free spaces around them; *Pach*, Pacchionian bodies surrounded by cancer cells and partly infiltrated by them (the left one); *V*, a vessel also surrounded by a focus of cancer cells, filled with blood, shows no carcinomatous elements within.

The pia-arachnoid studied with the same methods, also appeared alveolar-areolar in structure, the distended meshes packed with a great number of cancer cells (Fig. 3), many of which showed mitotic figures. Some contained more than one nucleus which frequently was biscuit shaped or horse-shoe shaped. The cells were usually scattered singly, but sometimes they were,

as in the dura, blended into giant cells or formed distinct foci. Many cells exhibited vacuoles, mostly filled with pigment granules, which were also frequently found over the connective tissue stroma. Other cells much resembled so-called macrophages, were of larger size and contained several distinct cysts with a dislocated flattened nucleus pushed to the periphery. Some cells were very pale, their cytoplasm converted into one large vacuole, the edges torn or broken up, the nucleus hardly visible. Cells were also encountered without any traces of a nucleus. While some of such cell fragments may be looked on as the result of cutting the sections, that is, have been divided by the knife, others may be considered as degenerated or disintegrated cell bodies.

Fig. 2.—Surface (horizontal) section of the dura. The interspaces between the bundles of connective tissue fibers are packed with cancer cells. Free spaces between the latter and the walls are clearly seen. Magnification 120 diameters. Van Gieson stain.

In addition to these degenerated or broken-up cells and macrophages, there could be seen a great number of "gitter cells" and plasma cells. The latter were freely scattered in the meshes of the connective tissue and could easily be recognized from their characteristic nucleus with its spoke-like arrangement of the chromatin, its excentric location and metachromatic cytoplasm frequently containing a halo. These cells were of unusually large size and often contained two nuclei.

The pia vessels were not proliferated, neither were they distended or infiltrated, but occasionally were thrombosed. Their walls, especially the

adventitia, were thickened but without any distended perivascular lymph spaces of Virchow-Robin. The connective tissue surrounding the vessels and elsewhere showed no fibroblasts, no exudation, the only pathologic phenomena, aside from the cancer cells, having been the macrophages, plasma cells and "gitter" cells. Though the last three forms of cells occur in various forms of acute meningitis, yet the absence in this case of vascular phenomena would make the diagnosis of carcinomatous meningitis rather problematic. On the whole, the pia changes were similar to those of the dura where the reactive phenomena were even less pronounced than in the pia; for instance, there was total absence of plasma cells.

The examination of the brain substance proper revealed an abundance of vessels and capillaries with densely stained endothelium and many so-called rod-like cells ("Stäbchenzellen") in the adventitial layer, but total absence of perivascular infiltration (Fig. 3).

The ganglion cells were greatly swollen, bottle or pear shaped, showed no Nissl bodies; their cytoplasm was lattice-like, or reticular in appearance, the processes well stained, and the nucleus pale and dislocated. Some cells were disintegrated and invaded by glia cells; that is, showed the phenomenon of neuronphagia. The glia cells were markedly proliferated, poor in protoplasm and contained a pale network of chromatin. In the cortical and subcortical areas the glia was distinctly reticular in appearance, showed a glia reticulum, and in some sections the brain tissue exhibited large perivascular and pericellular spaces, giving the section the appearance of so-called *état criblé*. Nowhere could any cancer cells be found in the brain tissue. Some portions of the brain, as the motor area, presented only glia changes, and slight parenchymatous or ganglion cell changes, while in other portions both were pronounced.

Of the cranial nerves the left third and both sixth nerves were examined with hematoxylin-eosin, Van Gieson and Alzheimer-Mann methods, and found practically normal, but the pia arachnoid around them was found slightly infiltrated, and, like the corresponding membranes over the cortex, showed complete absence of vascular changes.

In fact, all the membranes, including the dura, in spite of excessive cancerous infiltration (Fig. 3) behaved rather passively. The invaders multiply in their meshes and tissue spaces, but soon perish, becoming converted into lipoid substances or forming necrotic masses. There was some phagocytic activity displayed in the pia by the macrophages which pick up the broken up elements converting them into fat-like substances which subsequently are carried away by the "gitter" cells. Yet a diagnosis of meningitis, in the absence of vascular changes, is hardly justified. On the other hand, the brain substance proper exhibited marked parenchymatous changes combined with proliferation of the capillaries, of the glia nuclei and, what is more significant, showed a definite so-called glia reticulum which should be looked on, in my opinion, as specific for encephalitis.[22] The encephalitis would, perhaps, account for various nerve symptoms such as paralysis and

22. Hassin, G. B., and Bloch, L.: Trichnosis Complicated by Encephalitis, Med. Rec. **91**:537 (March 17) 1917.

anesthesias, which may be caused by the absorption of toxins produced by the cancer cells (Oppenheim[11]) or by the absorption of necrotic masses.

The cancerous infiltration of the subdural and subarachnoid spaces speaks for a connection existing between these spaces and the periphery; in the case under discussion, with the infiltrated glands of the neck. That such connection actually exists, was proved experimentally as long as fifty years ago by Key and Retzius,[23] and since then by numerous other workers. It would be proper to inquire how these

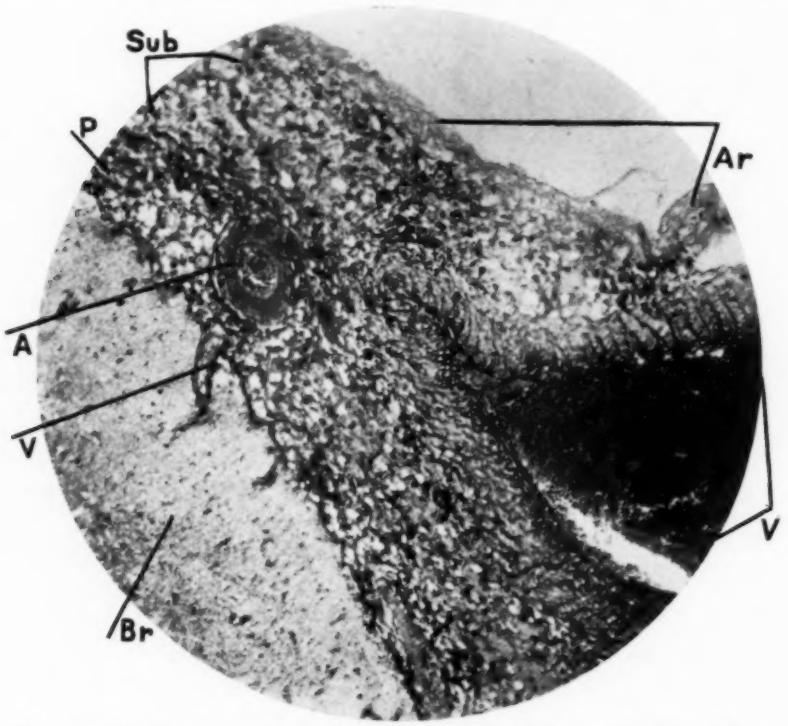


Fig. 3.—Transverse section through the arachnoid (*Ar*), Pia (*P*) membranes, subarachnoid (*Sub*) space, and brain (*Br*) tissue. *V*, a large thrombosed vein; *A*, artery; *v*, a smaller vessel given up by the pia. All the vessels show lack of perivascular infiltration. The numerous small black spots in the brain substance are proliferated capillaries. Magnification 80 diameters. Toluidin blue stain.

elements reached the various structures of the meninges; what pathways they had travelled from the periphery and within the meninges themselves. This problem has not been touched in the numerous

23. Key and Retzius: (a) Abstr. in Centralbl. f. med. Wissensch., 1871, p. 514; (b) Studien in der Anatomie des Nervensystems und des Bindegewebes **1**: 1875.

reports of carcinoma of the nervous system, except the brief assertion of Kaufman[7] that the meninges are reached by metastases through the lymphatics. If the cerebral membranes and the brain tissue possessed lymphatics, the mode of cancer cell transport to the nervous system would be quite simple. But there are no lymph vessels in the central nervous system[24] through which the fluid from the brain and its membranes can be drained, yet it is done in the most effective manner. This question was recently ably taken up by Weed,[25] whose brilliant experiments conclusively show that the cerebrospinal fluid flows from the subdural or subarachnoid space down to the corresponding, so-called perineural spaces of some cranial nerves (olfactory, trigeminal, hypoglossus), from which points it reaches through the tissue spaces the lymph vessels of the neck. Weed thus fully confirmed the earliest findings and observations of Key and Retzius,[23] Michel,[26] Schwalbe,[27] Quincke[28] and others. "The fluid," he says, "thus reaches the true lymphatic vessel only outside of the dura and of the cranium."

Assuming that cancer cells were a substance injected into the lymphatics, we must picture to ourselves the same pathway, but in an opposite direction. The cancer cells would have to travel from the lymph glands of the neck through the lymph vessels, tissue spaces, perineural spaces and finally reach the subarachnoid space. Then we must assume a backward or a retrograde flow of the lymph current, namely, from the neck to the brain, as otherwise it would be utterly impossible to explain the transportation of the cancer cells from the periphery. Recklinghausen[29] repeatedly advocated such a mode of metastasis, naming it "retrograde transportation of the cancer cells, in spite of the valves."

In a similar manner, that is, by way of a backward flow of the lymph from the infected cervical glands, were the cancer cells carried also to the subdural space.

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24. Poirier: Lymphatics, English Edition, 1904, p. 263. Sabin, Florence R.: (a) Development of the Lymphatic System; in Keibel-Mall's Manual of Human Embryology, **2**: 1912; (b) Harvey Lectures, 1915-1916, Series 11 (The Method of Growth of the Lymphatic System).

25. Weed, L. H.: (a) Studies on Cerebrospinal Fluid (Contributions II, III and IV, J. M. Res., 1914, No. 1; (b) An Anatomical Consideration of the Cerebrospinal Fluid, Anat. Rec. **12**: (May) 1917.

26. Michel: Berichte über die Verhandl. der Königl. Sächs. Gesellsch. d. Wissensch. z. Leipzig **24**:331, 1872.

27. Schwalbe, G.: Arch. f. mikrosk. Anat. **6**: 1870.

28. Quincke, H.: Zur Physiologie der Cerebrospinalen Flüssigkeit, Arch. f. Anat. u. Physiol., 1872, p. 152.

29 Recklinghausen: (a) Wien. klin. Wchnschr., 1897, No. 14; (b) Verhandl. d. deutsch. path. Gesellsch. **5**:167, 1903.

The latter, like the subarachnoid space, is present around each of the cranial nerves where they emerge from the cranium, running along them to various distances. In some nerves, for instance, the hypoglossus, it follows the latter's smallest ramifications, even as far as the tongue. In other words, each cranial nerve on leaving the skull cavity receives a covering, a sheath, from the dura, beneath which the arachnoid membrane is represented by another covering. Between these two membranes is the subdural space following the course of the nerve (Key and Retzius). Sooner or later these two membranes become blended, forming the so-called perineural sheath. Any substance injected into the subdural space, like Richardson blue, prussian blue, etc., easily reaches the lymph glands of the neck. Invasion of these perineural spaces, as Sabin calls them, by cancer cells while they run in the infected region will of course result in contamination of their prolongations, the subdural or subarachnoid space. Each may become involved independently of the other, along their own channels, their prolongations in the neck. That this actually occurred in our case is probable because the dura was loosely attached to the pia-arachnoid, the latter was perfectly smooth and showed no adhesions, which would have been present if the cells had traveled from the subdural space to the arachnoid or vice versa. These two spaces are closed formations, absolutely separate and independent of each other. Neither Key and Retzius, nor Weed were able to show the existence of any connection between the subdural and subarachnoid spaces. The contents of the latter are the product of the choroid plexus or of the perivascular so-called lymph spaces, those of the subdural space probably the product of the mesothelial cells of the dura proper (Weed). These cavities do not pass anything through their linings, unless high pressure is used (as was probably the case in the experiments of Quincke, who admitted a possible passage of cinnabar from the subdural into the subarachnoid space).

Evidently the subdural space is connected with the so-called dural interspaces (Fig. 2). The latter are generally looked on as lymph spaces, which is denied by Sabin[24] and Weed,[25] but were successfully injected from the subdural and subarachnoid spaces by Key-Retzius, Michel and others. At any rate, whether tissue spaces or lymph spaces, they are in communication with the subdural space, as in no other way could the cells reach them. To assume that the contents of the interspaces grew from the mesothelial cells by which they are lined or from the cells that normally fill up these interspaces known as arachnoid cell nests (Weed), is illogical, as in our case, the cells of the interspaces are identical with those of the subdural space and are lying freely separated from their walls (Fig. 2). On the other hand,

the lacunae (Fig. 1) were always devoid of contents. In none of them, in spite of the most diligent search could a single cancer cell be found. They were also free of contents in the injection specimens of Key-Retzius and of Weed.[25-a]

Scattered near the lacunar region are so-called Pacchionian bodies (Fig. 1), or hypertrophied villi, as Weed calls them; formations through which the spinal fluid filters, thus forming one, probably the principle, avenue of escape of this fluid from the subarachnoid space. In some of my specimens, these bodies were surrounded by masses of cancer cells or necrotic foci, and even within the bodies themselves cancer cells could be seen, though in very small numbers (Fig. 1). This would speak for the correctness of the opinion that the villi or Pacchionian granulations serve the purpose of eliminating the contents of the subarachnoid and subdural spaces.

As noted in the foregoing, the brain tissue showed no cancer cells. In spite of an enormous vascularization, the adventitial spaces of Virchow-Robin and the perivascular spaces of His were totally free from cancer elements. The Virchow-Robin spaces were clearly defined and described by Robin,[30] in 1859, though briefly outlined by Virchow in 1851.[31] They are spaces between the media and adventitia, while the spaces of His are located between adventitia and the brain tissue.[32] When excessively developed they produce what Durand Fardel called *état criblé*.[33] The spaces of Virchow-Robin are connected with subarachnoid space, the His spaces with the epicerebral space, situated according to His between the brain tissue and the pia. While Virchow-Robin spaces are universally recognized as actually existing formations, the His spaces as well as the epicerebral space and the *état criblé* are looked on as artefacts. This question I have discussed elsewhere,[22] and here wish merely to note that in my case all these formations were devoid of contents and that my findings do not speak for or against the views of His and his followers. The absence of contents from the Virchow-Robin spaces, however, is of great significance. Their contents are drained by the subarachnoid space towards which they flow from the brain tissue. Were the current *away* from the subarachnoid space *towards* the brain, toward its lymph spaces, we should expect in my case the latter to be filled

30. Robin, Charles: Recherches sur quelques particularités de la structure des capillaires de l'encéphale, Jour. de la physiol. **2**:537, 1859.

31. Virchow, R:. Ueber die Erweiterung kleinerer Gefässe, Virchow's Arch. f. path. Anat. **3**:445, 1851.

32. His, W.: Ueber ein perivascular Canalsystem in den nervösen Centralorganen und über dessen Beziehungen zum Lymphsystem, Ztschr f. Wissensch. Zool. **15**:127, 1865.

33. Durand-Fardel: Les maladies des viellards, Paris, Ed. 2, 1874, p. 121.

with cancer cells. This fact is again in accord with the conclusions of Key-Retzius and Weed, that the contents of the subarachnoid space come from within the brain.

The few points outlined demonstrate the scientific value of cases like the one under discussion, in studying the connections of the large cerebral spaces with each other, with the periphery and with the brain tissue proper. These questions are not only of theoretical, but also of practical value, for instance, in helping properly to estimate the value of intraspinal arsphenamin therapy (Strauss[34]). The foregoing anatomic questions have been attracting the attention of the ablest investigators for the last fifty years, and their solution was attempted by means of injection of various coloring substances (Richardson blue, prussian blue, cinnabar, tripan blue, fuchsin, plain mercury). In this case the injection was, as it were, done with cancer cells, and not by hands, but by nature herself, and must, therefore, be considered a much superior experiment and of greater scientific value.

In conclusion I wish to note that of the numerous staining methods used the most valuable proved to be Herxheimer's method of scarlet red, which allows of easy tracing of the epithelial, namely, the cancer cells loaded with red granules, along their various courses.

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34. Kaliski, D., and Strauss, I.: On Syphilis of the Nervous System, Am. J. Syphilis **2**:609 (Oct.) 1918.

# A CASE OF MONGOLISM IN ONE OF TWINS*

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The case we wish to report is the occurrence of Mongolian imbecility in one of twins, the normal child being distinctly above the average in intelligence. Since twins are subject to identical conditions from conception to birth, and usually enjoy practically the same environment until adolescence, the rare occurrence of Mongolism in one of them permits certain inferences relative to the etiology of this form of feeblemindedness. A brief résumé of the more pertinent facts concerning Mongolian feeblemindedness is appended, together with a review of the literature of Mongolism in twins.

## RÉSUMÉ OF MONGOLISM

One of the most clearly defined and best known types of feeblemindedness is that first designated as the Mongolian, Kalmuc or Tartar variety by J. D. Langdon Down[1] in 1866, so called from their facial resemblance to members of the Mongolian race. Tredgold[2] states that with the possible exception of the tongue, there are no physical signs that are pathognomonic of the condition, for all of the other anomalies present are seen in other types of feeblemindedness. However, it is the particular combination of anomalies which makes the Mongolian type so distinctive; those most important and frequently present being the small brachycephalic skull; small stature; obliquely set eyelids (hence the name Mongolian); large transversely fissured tongue; short, broad hands and feet; stubby fingers and toes; peculiar shortening and incurving of little finger (Telford-Smith finger); large cleft between the great toe and the second toe; hypermobility of the joints; defective speech; dry, rough skin, especially coarse on the extremities, and poor circulation. They are happy, smiling, good-natured, amiable, active, imitative, easily managed, and nearly always imbeciles, therefore possessing a mental age which corresponds to normal children of between 3 and 7 years. Various authorities state that this group comprises from 0.9 to 10 per cent. of all feebleminded;

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* To economize space, the geneological table and one photograph are omitted from THE ARCHIVES. The complete article appears in the author's reprints.

1. Down, J. L.: Clinical Lectures and Reports, London Hospital, 1866, p. 259.
2. Tredgold, A. F.: Mental Deficiency, Ed. 2, 1914, p. 211.

however, the minimum figure is too low. Statistics of feebleminded institutions show that from 2 to 5 per cent. of all admissions are of the Mongolian type, and since these cases can easily be managed at home, they are probably more numerous than these figures indicate. Goddard[3] estimates there are between 200,000 and 300,000 feebleminded in the United States. If we accept these figures there are probably between 5,000 and 15,000 cases of Mongolian feeblemindedness in this country.

The etiology of at least two-thirds of the cases of feeblemindedness is hereditary, due to defective germ plasm, that is, one or both parents are feebleminded, or there is a definite history of feeblemindedness in the ancestry. Neuropathic heredity, injuries affecting the brain before or at the time of birth, traumatism to the brain in early childhood, and various pathologic conditions of the brain, as meningitis, cerebral hemorrhage, etc., during the early years of life, are the etiologic factors in the cases not covered by defective germ plasm. Mongolian feeblemindedness is an exception, however, in that the etiology is unknown. As a rule, Mongols occur in the better families, there being but the one defective which is usually the first or last child born. The opinion of virtually all who have studied the condition is that it is due to something which interferes with prenatal development, and that the adequate cause is to be sought in the condition of the mother during pregnancy. Either uterine exhaustion, whereby the mother is not able to bring the child to full development (as seen when the child is the first born, the mother being very young, or where the child is the last born in a large family, or when the mother is nearing the menopause at the time of birth of the child) or some severe physical or mental shock to the mother which may have temporarily interfered with the procreative function, seem the most tenable theories. Vital exhaustion of either or both parents is regarded by some as the etiologic factor.

## LITERATURE OF MONGOLISM IN TWINS

A careful review of the American and English and a partial review of the remaining literature pertaining to Mongolism in twins has been made. The following cases have been reported:

Hjorth[4] reports the occurrence of Mongolian feeblemindedness in twins, each presenting the specific characteristics. The father was a day laborer and was 41 years of age at the birth of the children, the mother 42. These children were the eighth and ninth in a family of

3. Goddard, H. H.: Reference Handbook of the Medical Sciences, Ed. 3, **6**:384, 1916.

4. Hjorth, Bodil: Nyt Tidsskrift for Abnormvaesenet, No. 9, 1906.

ten. This is the only case reported that we have been able to find of the occurrence of definite Mongolism in both twins.

McKee,[5] in reporting fourteen cases of Mongolism of which one was in twins, states only that "one baby was the smaller of twins." Nothing is mentioned concerning the other child, but it is evident that it was not a Mongol.

Shuttleworth[6] states, "Among my illustrations I have the pleasure of showing you two remarkably fine photographs of twin children (boy and girl) from Melbourne, one (boy) normal, the other (girl) a Mongolian imbecile, for which I am indebted to Dr. A. Jeffreys Wood of that city. . . . The case of twin pregnancy with offspring of different sex, the boy normal, the girl a Mongolian imbecile, is unique in my experience."

## AUTHORS' CASE

*Family History.*—The parents of our patient are both collegiate graduates and highly respectable people. The father, now 50 years of age, married at 26, but on account of very poor health at that time, had to give up a confining position to get out of doors. His condition was diagnosed as pleurisy, tubercular origin suspected, and a grave prognosis given. He took up farming in order to build up his health and continued at this for many years. At present he enjoys fair health and his wife describes him as being "tall, slim, very nervous and lacking vitality." The mother, at present 53 years of age, has always enjoyed good health. She married at 29. As a result of this marriage there have been five children (two being twins) and two miscarriages. The oldest child, a girl of 23 years, is in fair health; the next, a girl of 21, is in excellent health. Both girls are brilliant students in one of our large universities. The next child is a boy of 20, who has tuberculosis. At present he is farming as his father did when similarly ill. The next pregnancy resulted in a miscarriage. Following this the twins were born, now 17 years of age, one being normal, the other a Mongolian imbecile. Another miscarriage followed the birth of the twins.

We have been fortunate in securing an excellent geneology of both the paternal and maternal sides of this family. In nearly 200 known blood relatives on both sides, representing five generations for the paternal and four for the maternal side, no cases of feeblemindedness have been established, except the one under consideration. There is one case of insanity—a religious mania—which affected a sister of our child's grandmother on the maternal side. There is one case of alcoholism which occurred in the maternal grandfather. The only tuberculosis is on the paternal side. Our patient's father in all probability had tuberculosis, the brother has it, an uncle recovered from it and the great-grandmother died of it. The paternal grandfather and grandmother died at about 80 from senility. The maternal grandfather, who was alcoholic, died suddenly at 45 from "heart trouble," and the grandmother at 78 from "anemia." Two other instances of twinning are seen in the family, both on the maternal side. An uncle of our case was the father of twin boys who were born prematurely, living but a few hours. The great grand-

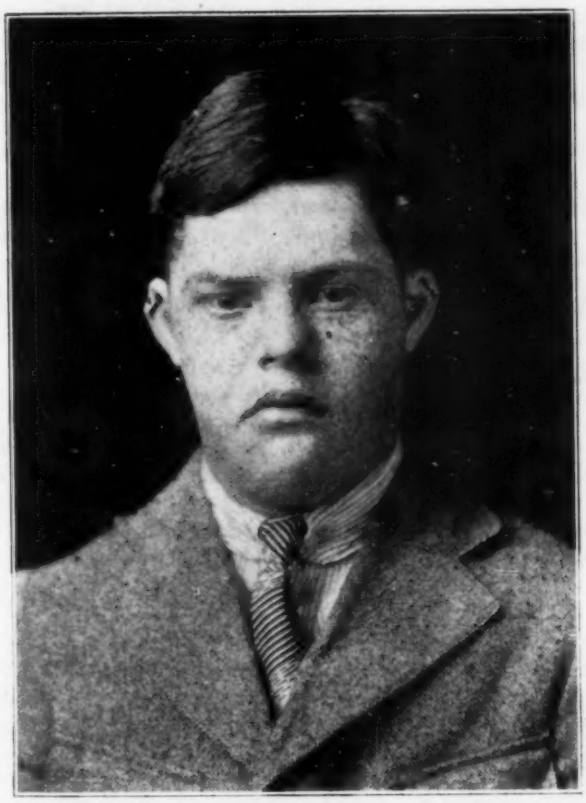
5. McKee, J. H.: J. Psycho-Asthenics **13**:43, 1908-1909.
6. Shuttleworth, G. E.: Brit. M. J. **2**:661 (Sept. 11) 1909.

father had brothers who were twins, and lived to old age. The brothers and sisters of our patient have had excellent school records and the majority of the relatives are above the average in intelligence, two uncles and one aunt being college professors.

*Mongolian Twin.—History.*—The patient was a boy, aged 17, whose father was 33 and mother 36 at the time of birth of twins who were the last born of a family of five. Mother had no unusual events during the pregnancy except that she frequently fainted. During previous pregnancies she was also subject to syncope, but with the twin gestation it was much more frequent. During the latter part of the pregnancy she suffered from severe pains in the upper part of the abdomen, presumably due to pressure from the fetal parts. The father was in fair health at the time of conception. The labor was normal, no instruments used, and the mother did not suffer more at this time than during her previous deliveries. The normal girl was the first born, weighing 5¾ pounds. She was a "blue baby" according to the mother, the "heart not closing" until the following day. The Mongolian boy was born a half hour after his normal sister. He appeared very pale, and weighed 6½ pounds. Even at birth he had the Mongolian facies, and the fingers were short and chubby in contrast to the long and slender ones of his twin sister. The integument remained soft and did not assume its normal firmness until 3 years of age. He was nursed by the mother and was a very good baby who seldom cried: Teething began at about 8 or 9 months, and was very irregular. He did not commence to walk until 3 years old and did not talk until 6. He never had any bad habits, and at present is quiet, good-natured and easily managed. At 2 years of age he had scarlet fever with otitis media as a complication which left a chronic discharging ear; at 3, measles and pertussis; and later adenoids which were removed. There is no history of convulsions. His schooling has consisted of two years at kindergarten, and three years of special class work for backward children in the public schools. From his 5th to 17th year, he was on thyroid treatment most of the time. The parents believe it has helped him and state his "tongue becomes thicker" and voice more imperfect when he is not taking the drug.

*Physical Examination.*—His height is 5 feet, and his weight 122 pounds. Musculature and nutrition are good. The head is somewhat small, but of normal shape, the greatest antero-posterior diameter being 6¾ inches, transverse diameter 5¼ inches, and circumference 20½ inches. The fontanelles are closed, the sutures are not palpable, the hair of the head is very abundant, coarse, straight and of red color. The face is freckled, normal symmetry, oval shape and covered with fine hair. The nose is short with nostrils tending to look forward. The upper part of the lobule of each ear is somewhat deformed but hearing is good. The eyes are slightly obliquely placed, the pupils are equal in size and react normally. The lids appear normal, no nystagmus or squint and vision is about 20/40 for each eye. The teeth are in fairly good condition, gums normal, palatal arch high and narrow, tonsils small, no adenoids and lips normal. The tongue is very large, and transversely fissured, papillae enlarged. The mouth is usually partially opened and the speech imperfect. There are no lymph glands palpable in the neck, the thyroid is not enlarged although the neck is large, the circumference being 14¾ inches. The respirations are regular, abdominal type and 20 in frequency. The lungs and heart are normal. The abdomen is protuberant with tendency to an umbilical hernia and there is a lordosis of the lumbar spine. The

genitalia are normal, with a normal amount of pubic hair which has a semi-feminine distribution. The hands and feet are of clumsy appearance. The fingers are short and stubby, but the thumbs and little fingers are not short, nor are the latter abnormally curved as often described as typical of Mongols. There is a large cleft between the great toe and its adjacent fellow. There is a great laxity of all the ligaments resulting in hyperextensibility of the joints. The skin is normal except that covering the hands which is thick and rough (not due to manual work). There are no varicose veins. The knee jerk and cremasteric reflexes are normal. The grip is good, gait normal, no paralyses



Authors' case. Mongolian imbecile, aged 17.

or tics, and coordination normal. The pulse is 74 and ocular pressure only lowers it to 69. The systolic blood pressure in the sitting posture is 135, diastolic 80. The temperature is normal and the hemoglobin, by the Tallqvist test, 80 per cent. A complete urinary analysis was not made, but the specimen was clear, straw color, characteristic odor, specific gravity 1.023 and no albumin present. The Wassermann reaction of the blood serum was negative.

*Psychologic Examination.*—(The psychologic examination was made by Miss Z. P. Buck, A.M., resident psychologist, Michigan Home and Training School.) The patient could not correctly repeat a sentence containing twelve syllables, failed to distinguish orange from yellow, did not know which was his left ear, could not describe pictures, could not repeat five digits, or repeat

three digits backward, could not count backward from twenty to one, could not give similarities between two simple things, could not define objects in terms superior to use, did not know day of week, month or year, could not discriminate between various weights, could not make simple change such as 4 from 10 or 12 from 15 cents, could not use three words in a sentence or give three words that would rhyme with given simple words, and his vocabulary was extremely poor. A more detailed psychologic examination was made and the following is the psychologist's report:

"By the Terman Test our case has a mental age of 6 years and 6 months, which gives him a retardation of 9 years and 6 months. The Seguin Board also gives him a mental age of between 6 and 7 years (Doll's Standardization).

"Throughout all tests the patient shows an ability to discriminate better than that of the average child of his mental age (Seguin, Card Dealing and Healey Picture Puzzles). His fatigue curve is irregular but remarkably well sustained as the curves show. It will be seen that even at the end of thirty minutes he is still making a higher record than during the first minute. The attention is, in general, poor. He is easily distracted and there is a limited amount of fluctuation. The powers of memory, apprehension, and apperception are also poor. In reasoning, with the exception of the comprehension question designed for a child of 8 years, and the most elementary addition problems, there is a complete failure. The will power in general is very good but will doubtless be used without reason. He responds immediately to praise and works with determination and thought such as he is capable of. The patient shows the usual Mongolian power of imitation. There is also a very evident personal pride. He carries himself erect and with chin up, frequently expands his chest to its greatest capacity and looks about for praise. He shows his muscles and readily undertakes anything that will increase his physical strength. Asked what he was doing in the cottage he straightened up, felt his muscle, and replied, 'growing'."

To summarize: The patient shows (1) good discrimination; (2) splendid endurance; (3) from fair to poor attention; (4) poor apprehension; (5) poor apperception; (6) poor memory; (7) very poor reasoning, and (8) a strong but irresponsible will power.

*Normal Twin.*—The twin of the Mongol is a normal, healthy girl. She has always enjoyed good health and in no way resembles her defective brother. She is 5 feet 5 inches tall and weighs 110 pounds. Her hands and feet are long and slender, her hair is fine and tongue normal. Both physically and mentally she has progressed as any normal child. She started in her school work at 7½ years of age. The first and second grades were completed in one year, as were also the sixth and seventh grades. She was graduated from high school at the age of 17. Her school record has been excellent and if we were to judge her mental ability by this, she could easily be placed above the average in intelligence. In her four years at high school, she never received less than 90 in any subject, and was always among the leaders of her class; her grand average for the four years being 93.4 per cent. Her best subject was mathematics, in which her average was 95.5 per cent. for the four years. This average included all the courses given in that subject.

## COMMENT

Many cases of twin births in the feebleminded have been reported. In most instances both children have been feebleminded, but in some cases one twin has been feebleminded while the other enjoyed a

normal mentality. Goddard[7] reports four such cases. This latter phenomenon can be easily understood if we accept Goddard's[8] explanation. He states that normal-mindedness is, or at least acts like, a unit character; is dominant and is transmitted in accordance with the Mendelian law of inheritance; feeblemindedness also behaves like a unit character, is recessive and is, like normal-mindedness, transmitted in Mendelian proportions. If feeblemindedness is transmitted according to Mendel's law, as there is very good reason to believe, it is easy to explain the difference in the mentality of the twins. Goddard[9] states: "We have two ova, fertilized by different spermatozoa, each of them subject to whatever possibilities the conditions of the chromosomes warrant. In the one case a "defective" spermatozoan has fertilized a "defective" ovum with the resulting defective offspring. In the other case a normal spermatozoan has fertilized a normal ovum, or else one of the germ cells has been normal and the other "defective," in either case resulting in a normal offspring."

However, the above discussion does not apply to Mongolian feeblemindedness, for it is an exception, in that it is not hereditary and does not, therefore, follow Mendel's laws. In this respect it is similar to those forms of feeblemindedness that are due to accidental causes acting before, at, or shortly after birth.

To our knowledge no Mongol has ever become a parent. The genital organs of Mongols appear to develop normally but the sex sense seems to be in abeyance, and some hold they are sterile. Their degree of mental deficiency is so pronounced that marriage is unthought of. This eliminates entirely the possibility of any light being thrown on the etiology by study of the offspring.

There are no grounds for explaining the condition on the basis of birth traumatism, for the physical characteristics of Mongols are already formed before birth. Moreover, in our case the birth was not only normal in every way, no instruments being used, but the normal twin was the first born and would, therefore, be the most likely to suffer from birth traumatism due to the crowded pelvis.

The mother suffered more during this pregnancy than any other, but this is to be expected in twin gestation. In twin pregnancies the mother is more subject to toxic conditions, etc., than when but one fetus is present.

As to the father's health at the time of conception little can be said. He was not robust, but was in much better physical condition

7. Goddard, H. H.: Feeble-Mindedness: Its Causes and Consequences, Macmillan Co., New York, 1914, p. 525.
8. Goddard, H. H.: Ibid., p. 548.
9. Goddard, H. H.: Ibid., p. 526.

than at the time of conception of his first child, when he was in all probability tubercular. As both parents are total abstainers, any thought of alcoholism can be eliminated.

Recently there has been an endeavor to explain the etiology of Mongolism on the grounds of congenital syphilis. As a result of serologic tests in thirty-eight Mongols, Stevens[10, 11] reports a positive Wassermann of the blood serum in 21 per cent., positive Wassermann of the spinal fluid in 18.4 per cent., and in 94.7 per cent. a positive reaction to Lange's gold chlorid test. He states that, "the serologic tests seem to demonstrate beyond question the condition is a result of syphilitic infection." McClelland and Ruh[12] answer Stevens' contentions by criticizing his method of interpreting the tests under discussion. In serologic tests of thirteen Mongols they found negative Wassermann reactions in the blood and spinal fluid and negative Lange's gold chlorid tests in every instance. They state, "from careful anamneses, physical examinations and the laboratory tests now available, it cannot be stated at the present time that Mongolism is due to congenital syphilis." Goddard[13] states that of twenty-eight Mongolians examined at the Columbus Institute, Ohio, 17.8 per cent. gave a positive Wassermann reaction (presumably of the blood serum); and at the Keller Institution in Denmark, no Mongolians gave a positive Wassermann reaction. Of his own cases at Vineland, where two series of examinations were made, and both blood and spinal fluid tested, he states, "in neither of these was the percentage of positives noticeably high." Quoting further from Goddard:

> "I have not been able to find any conclusive proof that syphilis in the parents causes feeblemindedness of any type, to say nothing of Mongolism. . . . To these difficulties in the way of regarding syphilis as the cause of Mongolian imbecility, I think may be properly added the further one that there is no correlation between the instance of Mongolism and syphilis. Mongolian imbecility is, relatively speaking, very rare; syphilis is far from rare. If syphilis is the cause of Mongolian imbecility, are we not compelled to conclude that the latter would be vastly more prevalent than it is?"

The only suggestion of syphilis in our case is the history of miscarriage before and after the birth of the twins. An examination of the blood serum of the defective twin gives a negative Wassermann.

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10. Stevens, H. C.: Mongolian Idiocy and Syphilis, J. A. M. A. **64**:1636, (May 15) 1915.

11. Stevens, H. C.: The Spinal Fluid in Mongolian Idiocy, Ibid. **66**:1373, (April 29) 1916.

12. McClelland, J. E., and Ruh, H. O.: Syphilis as a Factor in Mongolian Idiocy, J. A. M. A. **68**:777 (March 10) 1917.

13. Goddard, H. H.: Syphilis as an Etiologic Factor in Mongolian Idiocy, Ibid. **68**:1057 (April 7) 1917.

While it is true, syphilis would explain why one child was affected and not the other, the possibility of syphilis explaining all cases of Mongolism is very remote as the work of McClelland, Ruh and Goddard, quoted above, would indicate.

In conclusion, we may state that there is apparently nothing in the history of the mother, father or family to account for the occurrence of Mongolian imbecility in this twin. The defective child had the stigmata of the condition at birth; hence it is, no doubt, due to some congenital factor. Mongolism occurring in one of twins only further convinces us of the obscure etiology of this affliction. Granted, that it is due to an unknown congenital cause, we are at a loss to explain why one child is so affected and the other escapes entirely.

# EUNUCHOIDISM

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Castration was probably the first surgical operation. If that be correct, then the first surgical operation was not done to relieve human suffering but to satisfy one of the lower human instincts — jealousy. From the time of Semiramis to that of Abdul Hamid, eunuchs have been employed to guard the harems of the wealthy. Lipa Bey[1] vividly describes the barbaric way in which they are castrated and the tortures they underogo as children during and for a long time after this operation. Suffice it to say that according to Hirschfeld,[2] 80 per cent. of these unfortunates die either immediately during the process of operation or soon after from exhaustion due to pain and infection. Even today some of the children of the Orient are not free from the horrors of castration. In those parts of the East which are under the control of England stringent laws against this barbarism have been enacted.

It must, however, be admitted that even some of the ancient people protested rigidly against this form of brutality. The Hebrews considered castration an unpardonable sin. It was not lawful to castrate even animals. They went so far as not to allow a man whose sexual apparatus was cut off or otherwise mutilated to enter the house of the Lord.[3] We are informed, nevertheless, by that linguist and scholar in Semitics, Dr. Alexander Harkavey, that the Jewish kings employed foreign eunuchs in their harems. In fact, the Hebrew word "saris" had a double meaning. It is translated as eunuch and minister of court. Apparently the eunuchs occupied important positions in the courts of Jewish kings.

Human psychology is very strange indeed. We often inflict on ourselves brutalities in the name of ideals. In the eighteenth century

1. Bey, Lipa: Sexual Problem **7**:674, 1911.
2. Hirschfeld, M.: Sexual Problem, **8**:81, 1912.
3. Deuteronomy, XXIII, 11.

in Russia and in Roumania arose a Christian sect who thought that through castration they could enter with more ease the gates of Heaven. This sect called themselves Skoptzi in Russia and Lipowaner in Bukarest. They based their belief on the following passage from the New Testament:

> For there are some eunuchs which were so born from their mother's womb, and there are some eunuchs which were made eunuchs of men; and there be eunuchs which have made themselves eunuchs for the kingdom of heaven's sake. He that is able to receive it, let him receive it.—Matthew XIX, 12.

## LITERATURE

It was among the Skoptzi that Tandler and Gross[4] made their investigation which has since become classical. The importance of their studies consists mostly in the fact that acquired eunuchoism very closely resembles or, better still, is almost identical with eunuchoidism, the latter being a congenital condition.

Larey,[5] in "Campagne d'Egypte," was the first to describe a group of soldiers in Napoleon's army who had high pitched voices, fine skin, smooth and hairless, and no axillary or pubic hair. Their sexual organs were atrophied. For a long time these individuals were looked on as curiosities. Only a good deal later, when the study of the glands of internal secretions became more advanced, the pathology of this condition was taken up more seriously. Brissaud, de Santi, Levi, Anton[6] and others have shown this condition to be the result of a disturbed pluriglandular activity.

Pierre Marie, through his demonstration that acromegaly is due to pituitary disease, stimulated the study of this gland. Among other things it was shown that the hypophysis has control over the sexual glands. Later, Fröhlich[7] demonstrated his famous case of dystrophia adipositas genitalia which added a new asset to the knowledge of the pituitary. Soon after Cushing[8] demonstrated that the anterior lobe was responsible for giantism, hypoplasia of the genitalia and adiposity of the feminine type. Fränkel-Hochwart[9] demonstrated a patient in whom the power of erection returned after the excision of the pituitary gland. The literature soon became flooded with illustrations of the

4. Tandler, Julius, and Gross, Siegfried: Arch. f. Entwicklungsmechanik der Organismen **30**:236.

5. Larey, Baron: Quoted from W. Falta, Berl. klin. Wchnschr. **49**:1412, 1912.

6. Ibid.

7. Fröhlich, A.: Wien. klin. Rund. **15**: No. 47-48, 1901.

8. Cushing, Harvey: The Pituitary Body, J. B. Lippincott Co., Phila., 1911, p. 178.

9. Fränkel-Hochwart: Quoted from Peretz, Neurol. Centralbl. **29**:1286, 1910.

relation of the pituitary and sexual glands. McIver[10] reported a case of a giant eunuchoid, and ascribes it to hypopituitarism because the sella turcica is deep and the posterior clinoidal processes are well developed. Cocrayne[11] reported a young eunuchoid of the fatty type with genu valgum and also calls it hypopituitary. In Langmead's[12] case, one of mixed type of infantilism and dystrophia adipositas genitalia, the author finds the cause in deficiency of pituitary secretion.

Maranon and Pintor[13] report a case of eunuchoidism of the adiposita-genital type which developed following a bullet wound in the head. The necropsy, performed two years after the injury, showed an interruption of communication between the middle ventricle of the brain and the hypophysis. The hypophysis itself had undergone no direct lesion or compression. A case somewhat similar to this occurred in the Montefiore Hospital.

A girl, aged 11, with double optic atrophy, clinically showed symptoms of a pituitary tumor. The roentgenographic report was as follows: "The sella turcica is greatly enlarged; the clinoid processes are markedly thinned out and partly obliterated. Diagnosis: Neoplasm of the pituitary gland." The same report was issued on more than one occasion independently by two roentgenologists. The history was typical of increased intracranial pressure. At the beginning of the disease the child was markedly emaciated and the entire panniculus was wasted. A few months before her death she suddenly began to put on weight and in a short while she gained 40 pounds. She died from acute ascending paralysis. The necropsy showed a normal hypophysis and no tumor was found anywhere in the brain. Instead, a localized basal tubercular meningitis was present. This constricted the stalk of the hypophysis and thus interfered with its secretion. Symptoms of increased intracranial pressure were due to the internal hydrocephalus produced by the occlusion of the third ventricle. The rapid and enormous gain in weight can be explained by an interference with the proper function of the posterior lobe of the pituitary.

Manirot[14] reports a case of diabetes insipidus, general dystrophy and genital hypoplasia. He found a small sella turcica and attributes the symptoms to a diminution in the function of the hypophysis. Cases of eunuchoidism which resulted primarily from a disease of the pituitary are reported by Ebstein,[15] Weicksel[16] and Mattirolo.[17] St. Chauvet[18] discusses at length the subject of the relation of the pitui-

10. McIver, J.: J. Nerv. & Ment. Dis. **45**:443, 1917.
11. Cocrayne, E. A.: Brit. J. Child. Dis. **14**:52, 1917.
12. Langmead, F.: Brit. J. Child. Dis. **12**:241, 1915.
13. Maranon and Pintor: Nouv. Iconog. de la Salpêtrière, No. 4, 1917, p. 185.
14. Manirot, A.: Arch. de méd. des enfant **16**:927, 1913.
15. Ebstein, E.: Mittlg. a. d. Grenezgeb. d. Med. u. Chir. **25**:441, 1912-1913.
16. Weicksel, J.: München. med Wchnschr., No. 22, p. 1227, 1914.
17. Mattirolo, G.: Rêvista di Patol. Nerv: et Ment. **19**:513 (Sept.) 1914.
18. St. Chauvet: L'Infantilism Hypophysure, Thèse de Paris, 1914.

tary to eunuchoidism as well as to infantilism, while Mouriquand[19] divides the hypophyseal dystrophies into various types. Saenger[20] also speaks of the relation of the gonads to the pituitary. Contrary to established principle, as will be seen later, the fifth case of his series developed all the features of a eunuchoid in spite of the fact that the interstitial cells of the testicles were present. On the other hand, Fiessinger and Saurdel[21] report a case of gonadal atrophy associated with unmistakable signs of diminished thyroid and suprarenal functions. Rebattu and Gravier[22] report two eunuchoids, one with visible disturbance of the internal secretion and normal external secretion of the testicles, the other case with disturbed external and internal secretion of the testicles. They attempt to prove that the eunuchoidal characteristics of the individual depend on the absence of the cells of internal secretion in the testicle.

This condition was demonstrated experimentally by Ancel and Bouin,[23] who tied the blood vessels of the vas deferens in young animals, and although these afterward discharged normal spermatozoa, they acquired all the characteristics of castrated animals. One such case is reported in this article. Falta[24] discusses eunuchoidism acquired late in life either due to some disease or traumatism attacking the gonads. He tries to show that in these conditions we are dealing with a pluriglandular sclerosis.

Hecht[25] reports a case of eunuchoidism in an individual whose penis was very large, but whose testicles were of the size of lima beans. The patient had all of the other characteristics of a eunuchoid. He also showed levulosuria. Hecht comes to the conclusion that the condition is due to a pluriglandular disturbance.

Laignel-Lavastine and Courbon[26] report the case of a young soldier, 22 years of age, whose genital organs were normally developed until the age of 21. At that time he was stricken with mumps and a double orchitis. In the course of the next seven months the testicles and penis became markedly atrophied, the breasts increased in size, the abdomen became rounded, erection diminished, and sexual desire and power became weakened. Severe physical exercises in which he

19. Mouriquand, G.: Paris Medicale **6**:26, 1913.

20. Saenger, A.: Deutsch. Ztschr. f. Nervenh. **51**:178, 1914.

21. Fiessinger, Nöel, and Sourdel: Presse méd., No. 6, p. 541, 1911.

22. Rebattu, J., and Gravier, L.: Nouv. Iconog. de la Salpêtrière **26**:257, 1913.

23. Ancel, P., and Bouin, P.: Compt. rend. Acad. d. sc. **138**:110 (Feb.) 1904.

24. Falta, W.: Späteunochoidismus und Multipledrüssensklerose, Berl. klin. Wchnschr. **49**:1412.

25. Hecht, D.: J. Nerv. & Ment. Dis. **38**:684, 1911.

26. Laignel-Lavastine and Courbon, P.: Le Bulletin Med. **30**:408, 1917.

formerly excelled were no longer possible. Briefly, the individual presented a progressive transformation of the secondary sex characteristics into those of the opposite sex.

Butte and Halbeon[27] report a case of acquired eunuchoidism in a man 32 years of age. This was due to a syphilitic and gonorrheal infection at 20 which at 32 caused a general sclerosis of the glands of internal secretion. The patient became completely impotent, his testes atrophied, hair and teeth came out, he put on weight, and mentally he was a child. His blood pressure was low. With all that there was not a single sign of organic change in the cerebrospinal system. The authors attribute the syndrome to a sclerosis of all the glands of internal secretion.

Costa[28] reports a case of eunuchoidism with giantism in whom he could demonstrate a deficiency of all the glands of internal secretion. The author believes that the original cause was an infection of the testes in early childhood. A case of the same type is also reported by Schwaer.[29] Zambaco Pasha[30] claims that in eunuchoidism the gonads are not the only glands affected, but that the entire system of glands of internal secretion must be damaged in order to produce the clinical picture of the eunuchoid. Zambaco endeavors to show that each gland has its own definite function, but by damaging one gland all must necessarily be involved. Timme[31] expresses a similar view. Pende[32] reviews the subject of eunuchoidism and lays stress on the relation of the pituitary to the gonads. Goldstein[33] ascribes eunuchoidism to a pluriglandular disturbance. He, however, again repeats the hypothesis of Tandler and Gross that there is a mutually double interaction, inhibitory and acceleratory, between the hypophysis and the gonads. He believes that the anterior lobe, the one responsible for acromegaly, inhibits the sexual apparatus, while the posterior lobe accelerates it, that is, the two lobes are antagonistic to each other. In states of eunuchoidism the balance is lost and abnormalities in growth result which will depend on the predominating lobe. If the anterior lobe leads, there will be an abnormal growth of the skeleton; if the pos-

27. Butte and Halbeon: Bull. de mém. de la soc. méd. d. Hôp. de Paris **31**:365, 1915.

28. Costa, S.: Paris méd., No. 43, p. 378, 1913.

29. Schwaer, G.: Deutsch. med. Wchnschr. **40**:963, 1914.

30. Pasha, Zambaco: Les eunoques d'aujourd hui et ceux de jadis, Monograph, Paris, 1911, p. 152.

31. Timme, Walter: A New Polyglandular Compensatory Syndrome, Endocrinology **2**:209, 1918.

32. Pende, N.: Endocrinology **1**:924, 1916.

33. Goldstein, Kurt: Arch. f. Psych. u. Nervenh. **53**:649, 1914.

terior lobe leads we have a fat, bloated individual. This is a very attractive but as yet unproven theory.

More recently Ewing[34] has thrown new light on the subject. He epitomizes: "The fate of the human body is not a matter of chance, but in general is controlled by certain intrinsic physical tendencies that are of congenital origin." He teaches through necropsies that eunuchoidism is in many respects akin to status lymphaticus. In his postmortem reports of eunuchoids, Ewing especially emphasizes the hypoplasia of the cardiovascular system and a delicacy of all the blood vessels. This condition of the entire arterial system accounts for the frequent apoplexies in young individuals. The frequent interstitial myocarditis found at necropsies is responsible for the sudden death in these individuals.

A necropsy made by Dr. Harlow Brooks on a eunuchoid at the Montefiore Hospital in 1902 confirms Ewing's statement. We will repeat here a part of the necropsy findings.

> Large vessels of abdomen small and sclerotic (the individual, a boy, was 15 years old), especially the smaller trunks. Heart below normal in size, the weight being 9 ounces, with a slight amount of endocarditis. Thyroid small with an incomplete isthmus; weight 8 gm. Tuberculous lymph glands on neck. Liver small. *Spleen very large and asymmetrical. Adrenal bodies small and a portion of right adrenal lost.* The tissue has apparently undergone degeneration in the cortical portion. The kidneys are about one third of their normal size and show pathologic and congenital lobulation. Many cystic bodies are present. The genital organs are very small, a heavy fold of adipose tissue projecting over a very small penis 2 inches long, and covering mons veneris. The prostate is very small. The brain showed primitive sulci; it was small. Optic atrophy was present. Pituitary gland small, no gross lesions.

The individual was a eunuchoid of the fatty type.

More recently Citelli et Caliceti[35] report a number of young soldiers with feminine features who suffered mainly from adenoids. Caliceti[36] even goes so far as to demonstrate a case of pituitary disease associated with eunuchoidism cured by the removal of adenoids. He confirms the theory of Poppi who claims that all hypophyseal diseases are due to adenoid vegetations. As far as the gross pathology is concerned, particularly the skeleton, we cannot do better than to refer to Tandler and Gross,[37] and also Duckworth.[38]

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34. Ewing, James: Military Aspect of Status Lymphaticus, J. A. M. A. **71**:1525 (Nov. 9) 1918.

35. Citelli, S., and Caliceti, P.: Il Policlinico Lezione Pratica **25**:245, 1918.

36. Caliceti, P.: La Pediatria **25**:233, 1918.

37. Tandler, Julius, and Gross, Siegfried: Arch. f. u. Entwicklungsmechanik der Organismen **27**:35, 1909.

38. Duckworth, W. L. H.: J. Anat. & Physiol., 3d Series **2**:30.

DEFINITION OF THE TERM

Clinically, the term eunuchoid was first used by Duckworth and adopted by Tandler and Gross. The latter authors divide the eunuchoids into two classes: (1) tall, slender individuals with abnormally large extremities, and (2) stout, bloated people with accumulations of fat at the upper eyelids, breasts, lower abdomen, cristae iliae and

Fig. 1 (Case 1).—Eunuchoid, with spastic paraplegia due to apoplexy in cord.

nates. A number of intervening types are present. The symptomatology of eunuchoidism may be shown by a description of the following cases.

ILLUSTRATIVE CASES

CASE 1 (Fig. 1).—*History.*—A. H. entered the Montefiore Hospital Oct. 8, 1917. Father died at the age of 58 of cancer; seven brothers and sisters living

and well; one sister died in labor. Patient had been married six years and claimed to have had sexual intercourse. He, however, soon left his wife. Present illness dates back ten months prior to his entrance into the Montefiore Hospital. He complained of prickling sensations around the anus and right costal cartilage which lasted for a few days Suddenly at night he was stricken with paralysis of both lower extremities. He could not pass any urine and had to be catheterized for three weeks. His bladder condition gradually improved, and at time of examination he had perfect control of the urinary sphincter.

*Examination.*—The patient is 5 feet, 11 inches tall. Circumference of head, 20 inches. From acromion to styloid process of radius, 28 inches. Distance between anterior superior spines, 10 inches; anterior superior spine to internal malleolus, 34 inches; umbilicus to internal malleolus, 36½ inches; umbilicus to symphysis, 7 inches, and suprasternal notch to umbilicus, 12 inches. The patient has the typical appearance of a enunuchoid with pronounced features of feminism. There is an abundant growth of blond hair on the head, but the face is covered only with a whitish lanugo. The skin is smooth, soft, and velvety to the touch; it is of a pinkish hue with a tendency to flush. There is a profuse perspiration pouring down from axillae which are absolutely hairless. The abdomen is well rounded and shows a pigmented middle line. The pelvis is broad with a feminine contour. There is a perfect mons veneris covered with a few straight hairs which stop short in an almost straight line immediately above the mons veneris.

Genitalia: The penis is that of a child of 5 years. It measures 1½ inches from symphysis to meatus urethrae. The scrotum is markedly furrowed in the middle and as the patient lies recumbent it resembles the labia majora of the vulva. The testicles are very small, about the size of small gooseberries. When the scrotum is lifted the perineum is larger in size than the average man's perineum and has a tendency to shape itself into a fourchette. No hairs are to be seen on the perineum.

The lower limbs are feminine in type; they look as if they were chiselled of marble and have a fine pinkish hue. The breasts are well formed and are the size of those of an average girl at puberty. There are accumulations of fat at the lower abdomen and buttocks. The neck is perfectly round and the pomum adami is not visible. Voice is high pitched and feminine in character. Lower chin is somewhat protruded. Abdominal and thoracic viscera are negative.

The patient lies flat on his back being unable to stand or walk. He has a complete syndrome of spastic paraplegia; Babinski sign and other confirmatory great toe reflexes, double clonus, knee jerks and patellar reflexes are very lively. Cremasteric and lower abdominal reflexes not elicitable; upper abdominal reflexes present. While patient is being examined twitching of both lower extremities of the spinal epilepsy type is present. There is an area of hyperalgesia beginning at second lumbar vertebra and extending down to upper buttocks, where it becomes very marked. Here a prick of a pin throws both lower extremities into convulsions. Anteriorly there is a line of hyperalgesia at about the umbilicus and from Poupart's ligament down to the knee on left side and on the right side to the middle of the thigh. From here down to about the middle of legs sensation is intact, then there is anastomosis down to the toes. Analgesia follows the same distribution. There is a marked dissociation of temperature sense in the outlined areas. Vibration sense is absent in both lower extremities.

The upper extremities as well as the cranial nerves showed no abnormalities. There was no nystagmus and the disks were negative. The blood Wassermann test was negative. Cerebrospinal fluid taken on three occasions showed a normal pressure, clear, negative Wassermann, and globulin cells 6 to 8 to the field. The urine showed on one occasion granular and hyaline cells with some albumin; otherwise negative.

After giving 100 gm. of glucose in one hour doses, the blood showed 0.155 per cent. sugar. Urine was passed an hour after ingestion—quantity 160 c.c. and 1 per cent. of glucose. Twenty-four hours later no sugar was seen. The blood showed an average of lymphocyte count 25 per cent.; hemoglobin, 80 per cent.; red blood cells, 4,500,000; white blood cells, 10,000.

*Roentgenographic Report.*—Skull: spots of irregular bone condensation throughout the frontal and parietal bones; sella normal in size; hyperostosis of posterior clinoid processes. Marked diffuse atrophy in bones of feet. The lower extremities showed a moderately advanced bony rarefaction throughout the intratrochanteric portions of both femora.

*Subsequent History.*—The patient received practically no medication. He began to improve slowly and steadily and on April 28 we read the following bedside note made by the intern: "Complains of burning sensation in lower parts of legs. This sensation is especially distressing on prolonged walking." The motor power in his lower extremities markedly improved. In August he left the hospital to return to work. On September 29 he was again seen by one of us. He was working at his trade as a tailor. He still presented the spastic syndrome of paraplegia, but he was able to walk without any great difficulty. A marked improvement was also noticed in his sensory status. Otherwise the condition was unchanged.

*Mentality.*—The patient was a tailor and his mentality was that of the average tailor of his race and nativity. He could read Yiddish and understood some Hebrew. He spoke English not worse than the average Jewish tailor who has been in America a number of years, having been six years in New York. Simple questions in arithmetic were answered satisfactorily. As to his sexual status he felt a desire for women; he had married and had erections. He believes he had orgasms although there never were any ejaculations. When asked whether he would marry again, he smiled and stated that he had had quite enough of one wife. He is bashful and reserved, his nature soft and mellow. He absolutely refused to be demonstrated at a medical meeting. "God," he said, "has punished me enough. Neither you nor your colleagues can help me. Why should I expose my shame."

An analysis of this case shows that we are dealing with a eunuchoid with marked features of feminism. He is an intermediary type, being both tall and graceful and having the normal eunuchoid accumulation of fat. In spite of the roentgen ray report that the sella turcica is normal, he showed clinically distinct interference with the proper function of the pituitary gland. The lowered sugar tolerance as well as the accumulation of fat speak distinctly for a posterior lobe involvement, while the large and disproportionate skeleton together with hypoplasia of the sexual glands indicate an overactivity of the anterior lobe. The thyroid could not be palpated, but the profuse perspiration, the pronounced vasomotor change, evidenced both in flushes and dermographia, speak for an involvement of the thyroid and adrenals. We thus have a picture of pluriglandular disturbance.

The neurological syndrome can be explained only on the basis of a hemorrhage into the cord. The sudden onset of complete paraplegia followed by a gradual but steady improvement without medication, cannot be explained by any other condition except hemorrhage. As mentioned above, Ewing describes forms of apoplexies in eunuchoids and attributes them to an abnormally small heart and small walls of arteries. From the paresthesia that existed before the onset of the hemorrhage, we can conclude that there was a minute bleeding initiating the final hemorrhage. The present spasticity can be accounted for by secondary gliosis following the hemorrhage. Gliosis

Fig. 2 (Case 2).—Giant eunuchoid.

in eunuchoids was also reported by Burdels.[39] We therefore formulate our conclusion: The patient, a eunuchoid with marked feminism and pluriglandular disturbance, is suffering from a spinal gliosis following hemorrhage into the cord. After carefully searching the literature we could not find another case of apoplexy into the cord in eunuchoids, and came to the conclusion that this is probably the first case of its kind ever reported.

Case 2 (Fig. 2).—*History.*—D. S., aged 55, entered the Central and Neurological Hospital, Blackwell's Island, on Nov. 13, 1916. He complained of heart disease and inability to work. The patient is of a low grade mentality and no family history was obtainable beyond that he was born in Ireland and

39. Bartels, cited from Ewing.

his parents were farmers. He is a laborer, unmarried, and temperate in his habits, neither smoking nor drinking.

*Examination.*—The patient is fully 6 feet, 2 inches tall and of the appearance of a giant or atropoid. His head is small, 28 inches in circumference. As he stands he stoops somewhat forward. Double knock-knee, genu valgum, and flat feet. His hands are large and wide (Fig. 3) and spade-like in appearance. The face shows no growth of hair. The skin is yellow or parchment color and thrown into many folds and furrows. His forehead is narrow, lower chin protruded, eyebrows are thin and narrow. The skin over the body is thin and smooth. Chest is narrow with a tendency to pigeon breast. Thyroid

Fig. 3 (Case 2).—Genitalia and hands of eunuchoid.

not palpable. Pomum adami not visible. Distinct brown colored pigmentation over lower part of abdomen. Each pigment spot appears like a plaque and is the size of a lentil. The pelvis is broad, of the female type. Sergent's line is present.

The prostate was not palpable. His muscular power is greatly diminished. The muscles are flabby and appear atrophic. No electrical changes are found. His reflexes are sluggish, but no symptoms pointing to organic nervous disease is present. Wassermann test of the blood and cerebrospinal fluid is negative. The urine showed a few hyaline and granular casts with some albumin. One hundred gm. of sugar gave a distinct Fehling's reaction in the urine one hour after ingestion. Hemoglobin, 70 per cent.; red blood cells 4,000,000; white blood cells, 15,000; polymorphonuclears, 80 per cent. Some

of the ulcers on the legs were purulent. The heart shows a double systolic apical murmur. There was prolonged expiration and some dulness at both apices. The sputum was negative; blood pressure, 100. His voice was high pitched with some hoarseness. Unfortunately we could not secure an roentgenogram of his skull.

*Genitalia:* Distinct mons veneris covered with fine crisp gray hair. A small vermiform appendix that has the semblance of a penis and measures 1½ inches. The scrotum is mainly made up of adipose tissue. It is unproportionately large as compared with the penis. It is furrowed in the middle and resembles a vulva. Within each side of the scrotum is a small hard body, the size of a pea, probably a rudimentary testicle. Marked varicosity of lower extremities and ulcerations due to these varicosities.

*Mentality.*—He can neither read nor write, and is very poor in figures. He knows the days, but has some trouble with the months. In general his mentality is not above that of a child of 6 years. When asked as to his sexual feeling he looked blank and did not seem to comprehend the question well. He is obedient, reserved, not plaintive. When asked to stand before the camera there was no objection as is the case with so many of the eunuchoids.

This is a case of a giant eunuchoid with marked mental deficiency. The skeletal appearance is almost that of an acromegalic. It only differs from this condition in being congenital. Acromegaly is acquired later in life. This together with the low sugar tolerance would justify us in concluding that the pituitary is involved. The general weakness, the peculiar eruption, as well as the low tension, together with Sergent's white line, would speak of diminished adrenal function. That the adrenals are responsible for infantilism and eunuchoidism is also related by Morlat.[40] The testicles are certainly not functioning. If we now remember the close relation between the adrenals and thyroid we would say that the thyroid too must necessarily be involved. Therefore the case is the result of a pluriglandular disturbance.

A case of moderate imbecility associated with eunuchoidism where the libido was altogether absent is reported by Schwenkenbecher.[41] The patient was 17 years of age. Penis that of a child of 3, no testicles, no hair, small prostate, thyroid not palpable, small sella turcica, long extremities, separation of epiphysis. Moderate imbecility, kind nature, libido missing, no erections.

Case 3 (Fig. 4).—*History.*—M. S., aged 25, was born in Russia of Jewish parents. His father died of carcinoma of the stomach. *An older brother was not allowed to land in this country because of his low mentality. His*

40. Morlat, A.: Infantalisme et Insuffisance surrénale, Thèse, Paris, 1903. Quoted from B. Onuf. A Study of Eunuchoidism, etc. Am. J. Dermat. **16**:518, 1912.

41. Schwenkenbecher, A.: München. med. Wchnschr. **60**:444, 1913.

*genitalia were also undeveloped.* The patient is the third child, was born normally, breast fed, and had uneventful development until the age of 5. At that time he was stricken with scarlatina and says he was so sick that he could not speak for a number of weeks. At about the age of 10 he suffered from nasal obstruction. Soon after he had frequent urination and

Fig. 4 (Case 3).—Eunuchoid, status thymicolymphaticus.

enuresis. Frequent urination and nycturia, instead of enuresis, are still persistent. He attended a rabbinical school until the age of 12 and was of average brightness. At 18 it was brought to his attention that he had no desire for the opposite sex. It was also at that age that he first noticed that his sexual organs were not developed.

*Examination.*—He came to the Mt. Sinai Clinic complaining of "nervousness" and frequent urination and nycturia. He is 5 feet, 5 inches tall, and weighs 142 pounds. Circumference of chest at nipples, 34 inches. Circumference of head, 24 inches; upper arm 14½ inches, lower arm 10 inches. The hand measures 7 inches from the middle of a straight line thrown from the tip of the radius to the tip of the middle finger. Between outward superior 12 inches; from anterior superior to lower border of patella, 21½ inches; from there to internal malleolus, 15 inches. Length of foot, 11 inches.

Fig. 5 (Case 3).—Genitalia and thighs of eunuchoid.

As seen from figure 4, his form is that of a young woman. The breasts are well developed. The arms are round and well curved. The buttocks are round, hemispherical in shape. The thighs and legs and arms appear as if chiselled. The head dolichocephalic, thickly covered with jet black hair; face hairless, lower chin protruding, and on the perfectly round neck no pomum adami is visible; thyroid not palpable. The teeth are visibly separated and irregularly set. The posterior pharynx is full of adenoids; the breathing is

therefore through the mouth which is almost always slightly open. The hard palate is high arched. His voice is distinctly feminine with a nasal twang. The entire skin is smooth, soft and glossy. No hair on the axilla. The middle of abdomen is pigmented as seen in some women.

*Genitalia:* There is a perfectly well formed mons veneris covered with a few short and rarefied hairs ending abruptly at a straight line. The penis (Fig. 5), as well as the scrotum, is that of a child of 5 or 6. It is perfectly well formed, but measures only 1¾ inches. The scrotum is masculine in type but miniature in appearance.

*Clinical Examination.*—No heart lesion was found. There was some dulness present at upper manubrium; abdominal viscera negative; spleen and liver not palpable. Pulse 80 to 70. Blood pressure 110/70. Negative Wassermann reaction in blood and spinal fluid. Urine examination is negative. Blood Examination: Red blood cells, 4,000,000; white blood cells, 10,000; polymorphonuclear cells, 50 per cent.; small lymphocytes, 40 per cent.; large mononuclears, 4 per cent.; eosinophils, 4 per cent.; transitionals, 2 per cent.; hemoglobin, 80 per cent. No sugar was found in the urine following glucose given up to 400 gm. on an empty stomach.

*Roentgenographic Report* (Fig. 9).—Examination of the skull does not show the sella turcica very plainly but it appears somewhat flattened out and the clinoid processes seem to approach one another, if not almost touching. Examination of the chest shows a shadow at the root of the neck. Otherwise no abnormality is seen in the lungs, aside from marked enlargement of the bronchial lymph nodes.

*Mentality.*—His mentality is that of the average tailor. He is kind and soft natured. He takes part in the social movements of his class and belongs to some labor organizations. He obeys commands willingly. Has no objection to being demonstrated at medical meetings. He told one of us that he was released by the selective service draft board, but that he would rather fight and be a man than to be in his present condition.

This case is eunuchoid with a typical picture of status lymphaticus. Characteristic in this case is his family history. *A brother suffered from the same condition* which brought out the fact of this being a familial disease. This is mentioned by Ewing and many other writers on the subject. Unlike the findings of Tandler and Gross, Peretz[42] and many others, this patient has a *very small sella turcica.* He is one of those cases with congenital abnormalities which do not permit the hypophysis to expand in size. The roentgenogram of the chest suggests very plainly the possibility of a persistent thymus. This is still more emphasized by the adenoids and increased lymphatics in the bronchi as well as the rather increased number of lymphocytes in the blood. A. Müntzer[43] also speaks of observing a lymphocytosis in eunuchoids.

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42. Peretz: Refer to footnote 9.
43. Müntzer, A.: Berl. klin. Wchnschr. **472**:2052, 1910.

CASE 4.—*History.*—W. L., school boy, aged 15, admitted Nov. 9, 1916, to Mt. Sinai Hospital, service of Dr. B. Sachs. The chief complaint was obesity. Mother and father also obese; brother died of convulsions. Patient has always been a fat boy. Up to time of operation for appendectomy in 1913 his weight was 130 pounds and he now weighs 176 pounds. No cardiac, pulmonary or renal symptoms. Enuresis occasionally. He had measles in childhood.

*Examination.*—There is generalized obesity showing itself on face, chest, abdomen and extremities. Skin soft and white. No axillary hair. Head well formed. Neurological status, negative. Mouth negative except for markedly enlarged tonsils. Chest well formed; expansion good. Heart not enlarged, negative. Pulse equal; good force and tension. Abdomen: scar over appendicular region; no masses. Liver and spleen not palpable.

*Genitals:* Pubic hair scanty and ends in a straight line (feminine type). Small penis. Testicles very small, size of peas. Dilatation of internal mammary veins in right chest. Persistent thymus. Status thymicolymphaticus.

*Roentgenographic Report* (Nov. 14, 1916).—The skull shows rather large protruding frontal sinuses, large sphenoidal sinuses, and a marked prognatia of the jaws. At the same time the sella turcica is seen to be rather small. No other abnormaliy is seen in the skull. In the jaws the third lower molars are seen to be unerupted on both sides and are impacted against the second molars in a way which will interfere completely with their eruption. The third upper molars on both sides are also unerupted but they are not impacted.

## COMMENT

In this group of four cases we have a state of a congenital eunuchoidism, three of which, the first, third and fourth, can easily be put into the class of status lymphaticus, the first case giving clinical symptoms of apoplexy into the cord, a condition hitherto unreported, although apoplexies into the brain have been known to exist. The third case shows a sella condition that is also very rare and is contrary to the adopted opinion that in the eunuchoids the sella turcia is enlarged. It probably illustrates the fact that our judgment of the function of the pituitary must not be based on the size of the sella as evidenced through the roentgen ray appearance. The fourth case is a mixed type of eunuchoidism with characteristic fat accumulations and strongly suggestive of status lymphaticus. It will be noted that here also the sella turcica was rather small. The second case is in a class by itself. He resembles the anthropoid much more than does the average human being. His skeletal frame looks as if it belonged to an intermediary stage between the human and the anthropoid. His intelligence is low.

The question of the psychology of the eunuchoid has recently been studied by Sterling,[44] who divides the eunuchoids into three

44. Sterling, W.: Klin. Stud. u. Den Eunuchoidism, Ztschr. f. d. Gesam. Neurol. u. Psych. **16**:235, 1913.

classes: (1) Moderately diminished mentality; (2) imbecility, and (3) parasitic type. The first, third and fourth of our cases are, as regards mentality, normal beings anxious to be useful to themselves and others around them. Our case of imbecilism shows also an inversion to the lower stages in the development of the human race.

One thing can be said about the eunuchoids; all of them lack the masculine earmarks of psychology. They are conspicuous for lack of pugnacity, for their softness of character, weakness, and seeking the protection of others as do women. On the other hand, they lack

Fig. 6 (Case 5).—Acromegalic features.

the attractive part of woman's psychology. If we remember Virchow's aphorism that a woman is only a woman because she has ovaries, we must in our study of eunuchoids admit that whatever is masculine in a man is because of his testicles. Onuf,[45] who studied the eunuchoids from the standpoint of etiology and classification, also goes into the sociologic aspect, but arrives at no positive conclusion as to the social standard of this class of people.

Case 5 (Figs. 6 and 7).—*History.*—R. H., a woman, aged 22, was admitted to the Montefiore Hospital, June 16, 1918. The chief complaint was headache. The family history was negative except for a neurotic taint. The patient is

45. Onuf, B.: A Study of Eunuchoidism, etc., Am. J. Dermat. **16**:459, 1912.

married, but has no children. Menstruation began at 14 years, was regular for about six years when it ceased completely. She has had an abscess on the neck, but previously no serious illness.

The present illness which brought her to the hospital began about three years ago with headache localized over the right eye and right ear. For half a year the headache was not so severe, but after that it continued day and night. She never vomited. She had no trouble with eyesight, but believed that hearing on the right side was poor; no buzzing in the ears. Appetite fair, bowels constipated. No pulmonary, cardiac or renal symptoms. Configuration of face changed in one year.

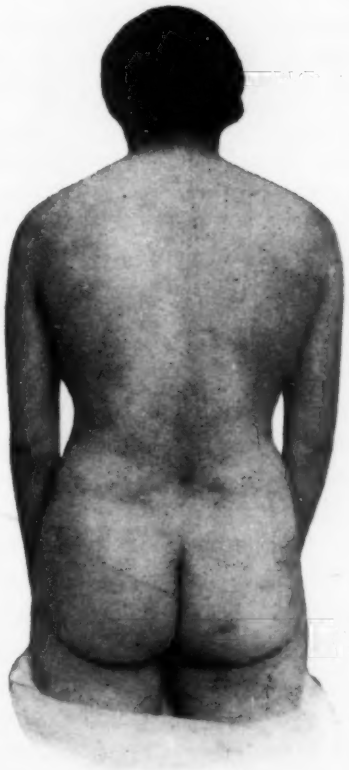

Fig. 7 (Case 5).—Posterior aspect—patient acquiring male shape of buttocks.

Patient noticed that the growth of hair on her legs and lower abdomen increased within a year. Tonsils and adenoids were removed on two occasions, and in the spring of 1916 she had a nasal operation performed, thinking that these minor operations would relieve her headaches. However, no relief was noticed. (The relation of adenoids to pituitary disease has been emphasized above.) The nasal operation was a resection of the cartilaginous and bony septum. There are no pathologic conditions in the nose, but there is a remnant of adenoid tissue in the nasopharynx. The fundi are normal. Blood Examination: Hemoglobin, 75 per cent.; red blood cells, 4,500,000; white blood cells, 10,000; polymorphonuclear cells, 50 per cent.; lymphocytes, 43 per

cent.; eosinophils, 1 per cent.; large mononuclear cells, 1 per cent.; transitionals, 4 per cent.

*Physical Examination.*—The head and neck were negative. Face: acromegalic; prominent lower jaw, brows and nose; high cheek bones, pasty skin, receding forehead and dry hair. The eyes were normal. The ears were negative externally. Mouth: tongue coated, teeth poor. Cranial nerves normal except a diminished sense of smell. Thyroid not enlarged. Glands: small inguinals palpable. Chest: expansion good; lungs negative anterior and posterior; heart: apex in sixth space, good force and quality, no thrills, action good, no murmurs. Abdomen soft and tympanitic, no masses; slight tenderness over the appendix.

*Genitals.*—Male distribution of pubic hair; uterus retroverted and retroflexed, small and atrophic. Extremities: Lower limbs: knee jerks and Achilles tendon reflex were present; no clonus, Babinski or Kernig signs; wide distribution of hair over entire legs. Upper limbs: wide distribution of hair on arms; hands have beginning acromegalic appearance, wide fingers and thick skin. The diagnosis of hypophyseal tumor was made and operation was advised. The urinalysis was negative except for diminished sugar tolerance. The blood Wassermann reaction was negative. The breasts were atrophied.

*Roentgenographic Report.*—Examination of the head shows a very large sella turcica with marked thinning of the anterior and posterior clinoid processes. The jaw is somewhat protruded, but there is no evidence of increased thickness in the bones of the skull. The frontal sinuses are entirely obliterated. The appearance is that of a tumor of the hypophysis.

This case is practically a eunuchoid of the female type developed subsequently to an involvement of the pituitary gland. The acromegalic features practically changed her countenance from a comely girl into the rude masculine type, with protruded eyebrows, well pronounced cheek bones, prognathism of the lower jaw and receding forehead. The color of the face changed from a blond and rosy hue into a dark color with a yellowish reflection. The skin lost all smooth and velvety feeling; it became more rough. Her buttocks have assumed a male contour. Her hands and feet have become large. The growth of hair that developed over her entire body completes the picture of inversion of secondary sexual characteristics. In other words, due to a diseased pituitary, a female has become a eunuchoid and has assumed masculine features.

Roberts,[46] in his travels in the surroundings of Bombay, observed women whose ovaries were cut out in early childhood. They too had assumed the masculine secondary characteristics. The mutual interrelation of the pituitary and sexual glands is once more illustrated. Our patient was practically castrated by nature interfering with the proper function of the pituitary gland.

46. Roberts: Quoted from St. Chauvet. L'Infantilisme hypophysure, Thèse de Paris, 1914.

CASE 6 (Fig. 8).—*History.*—This patient, L. B., aged 41, was born in Russia, and is a laborer by occupation. He married twice, having divorced his first wife because she had no children. He had mumps in early childhood. He came to the Mt. Sinai Clinic complaining of itching all over the skin.

Fig. 8 (Case 6).—Eunuchoid with large penis, but very small testicles, with some secondary feminine features.

*Examination.*—The patient is 5 feet, 10 inches in height, weighs 170 pounds, and is proportionately built. There is a fairly well grown moustache, and the face is wrinkled but not feminine. His breasts, however, are well developed and there is no hair on the body or in the axillae and the pubic hair is of the female type. The penis is normal in size. The scrotum is small in comparison with the penis. The testicles are the size of small gooseberries

and must be searched for within a scrotum that is much too large for such small testicles. The perineum is partly covered with hair. The prostate is very small. The contour of the buttocks is of the female type. The urinalysis is negative; 400 gm. of sugar give no glycosuria. The Wassermann test of the blood is negative. No neurological signs. Normal intelligence. Blood Examination: Red blood cells, 4,000,000; polymorphonuclear cells, 70 per cent.; small lymphocytes, 30 per cent.; large lymphocytes, 5 per cent.; transitionals, 2 per cent.; eosinophils, 1 per cent.; hemoglobin, 70 per cent. Blood pressure, 140/80. Voice high pitched. Seminal fluid shows a small number of monad

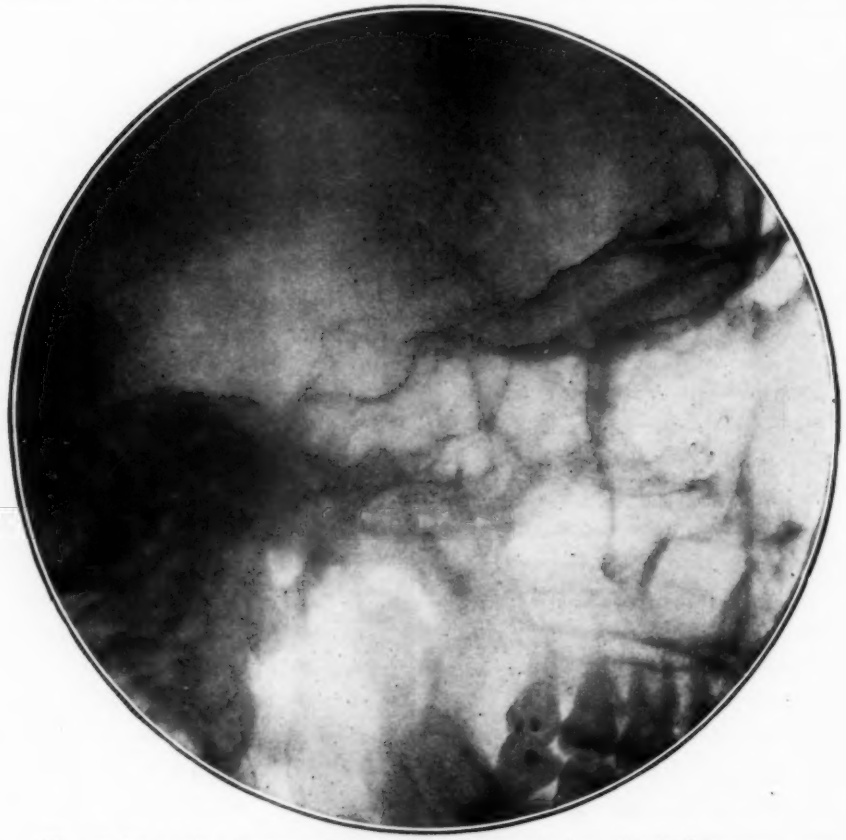

Fig. 9 (Case 6).—Large sella turcica with posterior clinoid processes overshadowing and concretions in the pineal gland.

cells, but not a trace of spermatozoa is found. He has indulged in coitus two and three times a week. While this patient has acquired some feminine features, in the main he is masculine in type.

*Roentgenographic Report* (Fig. 9).—Large sella with well developed and overshadowing posterior clinoid processes. Calcification of pineal gland.

## FUNCTIONS OF THE INTERNAL SECRETIONS

Reports of the relation between the pineal and sexual glands have been made by many reliable authorities. Bailey and Jelliffe[47] report

47. Bailey, P., and Jelliffe, S. E.: Arch. Int. Med. 8:851 (Dec.) 1911.

teratomas of pineal with sexual precocity in part and adiposo-genitalia syndrome. Fou[48] found that a removal of the pineal gland in cockerels caused a precocious development of the testicle and secondary sexual characteristics. Dana and Berkeley[49] found that the administration of pineal extract by mouth and intravenously is followed in young animals by a precocious development of the sexual glands. Tilney and Watson[50] showed that the pineal body is a gland *sui generis* and not a vestige of the third eye as it was maintained by Gaskel.

Fig. 10 (Case 3).—Sella turcica, flattened and proximal clinoid processes.

In Case 6 did the destruction or calcification of the pineal inhibit the testicular growth? We cannot assume any such deduction with any amount of certainty as we have seen through roentgen-ray examination so many calcified pineal glands without any clinical manifestations that it really cannot be considered as a sign of any value.

48. Fou: Quoted from E. A. Schäfer, "The Endocrine Organs."

49. Dana, C. L., and Berkeley, W. L.: Med. Rec. **83**:835, 1913.

50. Tilney, F., and Watson, L. F.: American Anatomical Memoirs, No. 9, Feb. 1919, Wistar Institute of Anatomy and Biology.

This case, however, illustrates another very valuable fact, namely, that the testicles have really a double secretion; an external, the procreative, and an internal which helps to make up the secondary sexual characteristics of the individual. In our patient the external secretion of the testicles was absent, yet he retained in main the secondary sexual characteristics and was able to indulge freely in coitus. The latter was observed by Tandler and Gross in some eunuchs in whom the testicles only were removed. All of our patients, except the imbecile, stated that they had a desire for the opposite sex. Lipa Bey[51] states that in the harem of Abdul Hamid a eunuch killed a young woman out of jealousy. It is, however, difficult to establish whether these people have a real libido.

One could probably interpret as a desire to be like others their apparent yearning for women. Our first case, though possessing only an infantile penis, even married. He wished to have a home. He left his wife, however, when she miscarried for he knew the child was not his. The high pitched voice is peculiar in all these cases. Scherer,[52] who studied this subject extensively, shows that in eunuchoids true laryngeal cartilages do not ossify, and that the bridge between the two thyroidal plates is much narrower than in men.

## TREATMENT

In Case 2 all sorts of opotherapy were used without results. It is almost needless to say that no treatment in these cases is of any avail. They are finished products and to improve or cure them would mean to create new tissues, an achievement as yet still to be looked for.

The subject of eunuchoidism is fascinating to those interested in the glands of internal secretion. The eunuchoid is *par excellence* a product of deficiency in the adjustment of the entire confederacy of these glands. It is in the eunuchoid that the key to a solution of some of the vital questions in endocrinology will be found.

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51. Lipa Bey: Refer to Footnote 1.

52. Scherer, Max: Ueber den Kehlkopf des Eunuchen, Berlin, 1901.

# THE PATHOGENESIS OF TABES DORSALIS

## WITH REMARKS ON TREATMENT OF TABES BASED ON CONSIDERATION OF PATHOLOGY *

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Although much has been written concerning the pathology of tabes, and particularly the causes underlying the degeneration of the posterior root fibers, writers are not yet in complete accord in their conception of the exact manner in which this degeneration takes place. Certain advances in our knowledge of this disease would warrant a reopening of the question. The importance of considering the disease as an active syphilitic one, the refinements of diagnosis by examination of the cerebrospinal fluid and the propriety of certain methods of local intraspinous medication would stimulate an inquiry into a more exact knowledge of the manner in which the spirocheta ultimately, and after a considerable lapse of time as a rule, causes a degeneration of the nervous parenchyma.

In 1917, I[1] published an article on the "Early Diagnosis of Tabes," and stated among other conclusions that: "In tabes the syphilitic posterior leptomeningitis of the spinal cord (and probably also of the radicular nerve) is in etiologic relationship to the degeneration of the posterior columns of the cord. Every case of syphilitic posterior leptomeningitis and consequent multiple symmetrical radiculitis is a case of potential tabes." These conclusions were based in part on a study of the necropsy findings in a number of cases of tabes dorsalis, which are further reviewed in tabulated form in this article. Before these cases are analyzed, and other considerations bearing on this subject are dealt with in more or less detail it would seem desirable to review the literature on the question of the pathogenesis of tabes.

### THEORIES OF TABES

Of these we may mention the older theories of primary gliosis, of vascular and of neuritic origin, the toxic theory first advanced by Strumpell (1882), the ganglionic theory of Oppenheim, the lymphatic theory of Marie-Guillian, the exhaustion theory of Edinger, the theories of a meningeal cause of the disease advanced by Obersteiner and

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* From the Neurological Clinic, Leland Stanford Junior University School of Medicine.

1. Schaller, W. F.: Early Diagnosis of Tabes Dorsalis, J. A. M. A. **68**:190 (Jan. 20) 1917.

Redlich, and by Nageotte, and recently the hypothesis of Orr and Rows of an ascending perineural infection. Of all these theories the meningeal theories and toxic theory occupy the foreground of attention.

It is now twenty-five years since the classic studies of Obersteiner and Redlich[2] and of Nageotte[3] first appeared. The publication of Obersteiner and Redlich precedes that of Nageotte by a short interval only. It may be stated at once that these two theories have much in common and are in fact, basically the same. Obersteiner states Lange was the first to affirm that spinal meningitis was the primary lesion of tabes. He asserts that the theory of Nageotte differs from his own only in that the posterior roots are affected in a different location: Obersteiner and Redlich hold that the posterior roots are compressed in their path through the pial ring formed by the spinal pia mater, and that this compression is due in part to an inflammation or shrinking of the meningeal covering and in part to pressure from sclerosed arterial vessels. That the retraction of the pia is always due to a meningitis is not asserted, as the assumption of a constant pial inflammation appeared to be the chief objection to this theory. These authors believe that this process may take place on an anatomic individual basis. They contend that the posterior root fibers show a more intense degeneration in their intramedullary course than in their extramedullary course.

Nageotte believes that posterior column degeneration is due to an inflammatory process of the radicular nerve ("nerf radiculaire") involving endoneurium as well as perineurium and taking the form of a transverse neuritis, starting as an inflammatory process of the meninges in this locality. The radicular nerve includes both the sensory and motor roots, and extends mesially from the spinal ganglion to the beginning of the dura and arachnoidal envelop, and is in very intimate relation with it. The more resistant motor fibers escape, the sensory do not. This area of the radicular nerve is also considered to be a very important avenue of communication between the cerebrospinal fluid and the perineural lymphatic spaces.

Soon after the above theories were advanced, Philippe[4] in France (1897), and Redlich[5] in Germany (1897) published comprehensive

2. Obersteiner and Redlich: Ueber Wesen und Pathogenese der täbischen Hinterstrangs Degeneration, Obersteiner's Arbeiten, 1894; Abstr. Neurol. Centralbl. **13**:454, 1894.

3. Nageotte, J.: Bulletins de la Société Anatomique de Paris, **8**: (5 série) 808, 1894.

4. Philippe, C.: Le Tabes Dorsalis, Bailliére et Fils., Paris, 1897.

5. Redlich, E.: Die Pathologie der täbischen Hinterstrangserkrankung. Gustav Fisher, Jena, 1897.

monographs on the pathology of tabes. A toxic element acting on the intramedullary portion of the posterior roots is mentioned by Redlich as a probable factor, and he also favors the hypothesis of Edinger. It thusly appears from this later publication of Redlich that he had modified his previous views to an extent. He also states that even mild forms of meningitis cannot always be demonstrated in tabes, although the meninges cannot be said to be entirely free from a slight "thickening" without particular significance.

Philippe also states that the posterior meningitis is too inconstant and too out of proportion to explain the tabetic degeneration as being an initial interstitial process. He quotes a case previously reported by himself, one of syringomyelia, in which although the meningeal lesions were extremely pronounced, they did not occasion a degeneration of the roots which traversed them. He concludes that the degeneration is primarily a parenchymatous one which causes a degeneration of fibers without first affecting the cells of origin.

At this period Spiller[6] made the important observation that tabes is to be considered rather as a cerebrospinal disease. In a later paper this writer[7] discusses the pathology of tabetic ocular palsy. In reference to cerebrospinal syphilis he states that in his experiences round cell infiltration, lymphocytic infiltration of the pia, is the most characteristic lesion of cerebrospinal syphilis, and that no acute manifestation of syphilis of the cerebrospinal axis exists without it. He reports a case of complete bilateral ophthalmoplegia in a tabetic and from a study of the abducens nerves found evidence that the lesion was peripheral, that is, in the nerve itself. This would be opposed to the prevailing idea of the pathology of the ocular palsies of tabes and of brain syphilis in which in tabes the ocular paresis is due primarily to nuclear involvement, whereas in cerebrospinal syphilis it is due primarily to nerve involvement.

Derjerine,[8] in his book on "Disease of the Spinal Cord," in reviewing the different theories, believes that neither the posterior meningitis or radicular meningitis can explain, on an exclusively anatomic basis, the degeneration of the posterior roots. He, however, admits that the diffuse meningitis which exists is an expression of an intoxication or toxi-infection of the sub-arachnoid space, and in this manner serves to elucidate the pathogenesis of tabes. That tabes is due to a

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6. Spiller, W. G.: The Pathology of Tabes Dorsalis, Int. Med. Mag. **5**: No. 5, 1897.

7. Spiller, W. G.: Pathology of Tabetic Ocular Palsy, etc., J. Nerv. & Ment. Dis. **42**:15, 1915.

8. Derjerine and Thomas: Traité des Maladies de la Moelle Epinière, Paris. J.-B. Bailliére et Fils, 1902.

primary posterior root involvement is strongly maintained by this authority. The intramedullary degeneration, the sparing of the endogenous sensory fibers, and the presence of the peculiar degeneration in early tabes in Burdach's column first described by Pierret (1877) are all in favor of this manner of viewing the problem. Derjerine compares the bandelette externe of Pierret with its central extension in Goll's column forming a figure likened to an M, to the middle radicular zone of Flechsig; the degeneration is therefore systematized and contains medium and short fibers of the posterior roots.

Rhein[9] has studied the lesion of the radicular nerve as described by Nageotte, and also examined the spinal meninges in a considerable number of cases of tabes. He concludes that the radicular nerve lesion is not constant, and that meningitis may exist without causing the characteristic cord lesions in tabes. Strumpell,[10] in a recent communication, still maintains that the essential cause of the posterior column degeneration is toxic, and in this view he is supported by Sepp,[11] who quotes Tinel on the toxic action of meningeal exudates. Karl Schaffer[12] in Lewandowsky's system describes the condition as a primary elective fiber degeneration. Other adherents to the toxic theory are Marinesco and Minea;[13] Austregesilo.[14] A considerable support to the supporters of the toxic theory has been given in the recent experiments of Spielmeyer.[15] This author was struck by the similarity in symptoms between general paralysis and sleeping sickness, and conceived the idea of infecting animals with the *Trypanosoma brucei*. For this purpose he chose a degenerated race of dogs—spitz dogs and poodles. After keeping these severely infected animals alive for a period of nine or ten weeks a degeneration was found by Marchi's method in the posterior spinal roots, the spinal root of the fifth cranial nerve and in the optic nerve, the tabes being of the cervical type. No change was found in the ganglion cells, the spinal pia or the peripheral

9. Rhein, J. H. W.: Meningitis and Disease of the Radicular Nerves in Tabes Dorsalis, Univ. of Penn. Contributions, Neurology, No 5, 1909-1910.

10. Strümpell, A. v.: Pathologie und Behandlung der Tabes Dorsalis, Wien. med. Wchnschr. **61**:1214, 1911.

11. Sepp: Ueber die Pathogenese der Tabes, Deutsch. Ztschr. f. Nervenh. **52**:46, 1914.

12. Schaffer, K.: Tabes Dorsalis, Lewandowsky's System, Handbuch der Neurologie **2**:1916, Spezielle Neurologie 1.

13. Marinesco, G., and Minea, J.: Contribution a l'anatomie pathologique et a la pathogénie du tabes, J. f. Psychol. u. Neurol. **10**:182, 1907-1908.

14. Austregesilo, O.: Frühdiagnose der Tabes oligosymptomatica, Deutsch. med. Wchnschr. **39**:2396, 1913.

15. Spielmeyer, W.: Experimentelle Tabes bei Hunden (Trypanosomen-Tabes), München. med. Wchnschr. **53**:2338, 1906.

Lower dorsal region: Over a thickened pia, there is a superimposed lymphocytic meningitis. Note the perivascular cellular infiltration. Hematoxylin-Van Gieson.

nerves. The process described by Spielmeyer is an elective disease of the centripetal portion of the sensory protoneuron. This same investigator also studied experimental dourine in dogs where inflammatory meningeal processes took place, but no degenerative changes in the posterior roots. Root fiber disease in animals is therefore not dependent on infiltrative processes according to Spielmeyer.

In horses, however, in which this disease occurs, Mott[16] found both meningeal changes and marked posterior column involvement. The degenerations were so marked in one case that the degenerated areas could be seen macroscopically. Mott makes mention of the similarity between this pathologic picture and that presented by tabes. Clinically, also, there are points of resemblance, such as spontaneous fractures and dislocations which often occur in horses affected with this disease.

In a Rhesus monkey which was sent to him for experimental purposes, Rothmann[17] noted ataxia, diminished vision and disturbed cutaneous sensibility. After experimentation (pyramidal tract extirpation) examination of the cord showed posterior column degeneration in addition to degeneration in the pyramidal tracts. Because this degeneration was not accompanied by a pial meningitis, it was therefore considered to be primarily toxic. Schroder[18] reports an affection simulating tabes in a monkey (cercopithecus fulginosis). The histologic picture of the cord degeneration resembled the changes of pernicious anemia in man.

Spielmeyer[19] has studied cord degeneration produced by intraspinal stovain anesthesia. In a fatal human case the lesions were predominately in the cells of the anterior horns, but in experiments on dogs and monkeys this same investigator found the principal lesions in the posterior columns. In stovain tabes the extramedullary portion of the posterior roots are the ones first affected by the neurotoxic agent and the degeneration of the intramedullary portion of the roots is a consequence of the primary degeneration in the extramedullary portion. In this respect the degenerations differ from trypanosome tabes where the intramedullary portion of the root fibers are first affected, as is the case in ordinary tabes dorsalis. Therefore in stovain tabes the changes in the posterior roots are not due to a *locus minoris*

16. Mott, F. W.: Histological Observations on the Changes in the Nervous System in Trypanosome Infections, Especially Sleeping Sickness and Dourine, etc., Arch. Neurol. **3**:581, 1907.

17. Rothmann, M.: Ueber eine Tabes-Artiges Erkrankung beim Affen, Monatschr. f. Psychiat. u. Neurol. **20**:204, 1906.

18. Schroder, P.: Ueber eine Hinterstrang und Sehnervenerkrankung beim Affen, Arch. f. Psychiat. **44**:193, 1908.

19. Spielmeyer, W.: Veränderungen des Nervensystems nach Stovain Anästhesie, München. med. Wchnschr. **55**:1629, 1908.

*resistentiae* at the border line perforation of the pia. When the degenerations are extensive in the posterior columns other fiber systems are also involved and degenerations are noted in the periphery of the cord and in the anterior roots. The anterior horn cell degeneration in animals has never been observed in man. Spielmeyer concludes that the pathologic process is due to a direct toxic agent. Inflammatory reaction, sclerosis or hemorrhage are absent.

Mosse and Rothmann[20] have described cord lesions in dogs rendered anemic by subcutaneous injections of pyroiden. The degenerated areas in the cord are not confined to the posterior columns, but occur with almost the same intensity in the lateral and anterolateral columns.

Degeneration of the posterior cord columns very similar to that occurring in tabes is described by Tuczek[21] due to poisoning from ergot. In four necropsy cases Burdach's column was involved wholly or in part showing a symmetrical degeneration with a narrow intact strip between it and the posterior horn and the posterior commissure. According to Tuczek "ergot tabes" differ from tabes dorsalis only in the acute nature of the process. The symptomatology is also similar: absent lower tendon reflexes, Romberg, ataxia, paresthesias and pain. The permanent after-effects have been emphasized by Walker and Jahrmarker. Cases in literature are also referred to in which anatomic degenerations in the posterior columns have been demonstrated occurring more or less acutely in such conditions as scarlet fever, diphtheria, following the puerperium, .and from absinthe poisoning (Tuczek).

In such chronic or subacute affections as leukemia, the metal poisonings, notably lead poisoning, chronic alcoholism and nutritional disorders such as beri beri, posterior column changes have been described, but these are either associated with, or dependent on, peripheral nerve disease, and appear to throw no light on the tabetic process. Williamson[22] has studied the spinal lesions in diabetes and found degenerations in the intramedullary fibers of the posterior roots. He believes that these changes may occur without associated peripheral nerve disease, or at any rate without significant peripheral nerve disease.

The degenerations of pellagra also produce posterior column changes which were first studied by Borchard in 1864; later by Tuczek. The degenerations are of the combined system type. Neither vascular

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20. Mosse, M., and Rothmann, M.: Ueber Pyroidenvergiftungen bei Hunden, Deutsch. med. Wchnschr. **32**:134, 187, 1906.

21. Tuczek, F.: Ueber die Veranderungen im Centralnervensystem, spezialle in den Hinterstrangen bei Ergotismus, Arch. f. Psychiat. **13**:99, 1882.

22. Williamson, R. T.: Disease of the Spinal Cord, Oxford Univ. Press, London, 1908, p. 371.

or meningeal changes are found. Quite recently Wilson[23] in a material of thirteen cases has made a detailed pathologic study of the degenerations and states that the cord lesions are pseudosystematized resembling combined sclerosis. The absence of any inflammatory reaction is noteworthy.

Long,[24] however, in this disease has dwelt on the presence of cartilaginous-like deposits in the spinal foraminae demonstrated by roentgenographs and found also in one necropsy case. He explains the spinal root degeneration by root pressure. In support of this explanation anatomic considerations would appear to have a bearing, for in the lower cervical region the spinal foraminae are relatively smaller in relation to the nerve roots which traverse them than is the case in the upper cervical or dorsal regions, and this would explain the hand atrophies. Likewise, the degenerations of $S_1$ and $S_2$ are possibly explained by pressure paralysis of these roots passing through the tortuous and flattened foraminae in these localities.

### ANOTHER ASPECT OF THE PROBLEM

From the considerations of the foregoing meningeal and toxic theories and other conditions related to tabes which might throw some light on the pathogenesis, let us now turn to another aspect of the problem. It has been stated above that Spiller as far back as 1897 asserted that tabes is in reality a cerebro-spinal disease. I believe that this position has been justified by the advance in our knowledge of tabes which has come about by studies of cranial nerve lesions in this affection. Spiller's work on tabetic ocular palsy has been mentioned. Vincent,[25] in his Paris thesis, studied the degenerations of the third, ninth, eleventh and twelfth cranial nerves in tabes. He found, as did Nageotte in the spinal roots, a meningo-radiculitis where the cranial nerves passed through the dura and arachnoid coverings. From a careful study by serial sections of the nerves in this locality he concluded that the inflammatory process was intense enough to explain the cranial nerve degenerations.

In early syphilis, diminution of hearing has been explained by the presence of a specific leptomeningitis and consequent trochlear nerve involvement affecting the eighth nerve by Knick and Zalozieki,[26] and

23. Wilson, S. A. K.: Pathology of Pellagra, Proc. Roy. Soc. Med., London **7**:31, Neurol Sec., 1914.

24. Long, J. D.: Quoted by Niles, Pellagra, Ed. 2, W. B. Saunders Co., p. 159, 1916.

25. Vincent, C.: Des meningites chroniques syphilitiques, Thése de Paris, Paris, G. Steinheil, 1910.

26. Knick, H., and Zaloziecki, A.: Ueber Acusticuserkrankung im Frühstadium der Lues, Berl. klin. Wchnschr. **49**:639, 1912.

by Ellis and Swift.[27] Similarly the characteristic bilateral facial palsy has been explained due to early meningeal syphilitic involvement by a number of recent observers. Leri[28] has expressed the opinion that the optic atrophy in tabes is due to an interstitial process of the optic nerve itself due to syphilitic meningitis and syphilitic vascular disease. Schlagenhaufer,[29] in a study of the course of the optic nerve fibers in a case of tabetic optic atrophy, found lesions of the optic nerve trunk in the passage through the optic foraminae, and because of the complicated nature of the disease process regarded it as due to a periostitis syphilitica or pachymeningitis.

### OPTIC ATROPHY

The existence of a tabetic primary optic atrophy has also of late been seriously questioned by Stargardt[30] in a rather convincing study in the Kiel clinic. This study consisted of an examination of the optic nerves and retina in twenty-four cases of tabes, general paralysis, and tabo-paralysis, and nine other normal cases for control. By selected staining methods and particularly by early and suitable fixation of the retina this investigator has come to the conclusion that the optic atrophy in the foregoing disease is not due to a primary degenerative toxic process in the retina as has been assumed, but that it is due to an exudative process in the optic nerve itself chiefly in the bony canal, the intracranial portion of the nerve and the chiasma. Characteristic changes previously described in the retina are interpreted by Stargardt as due to postmortem changes from delayed fixation. He is very emphatic on the basis of his histopathologic studies that the primary lesion is in the optic nerve and not in the retina. The following pathologic changes were noted:

1. Changes described as exudative processes consisting of the presence of plasma cells and lymphocytes in pia and septum of optic nerve and in the perivascular lymph spaces. The changes are described as "infiltrative."

2. The presence of vascular changes such as described by Alzheimer (progressive form) in the localities affected by exudative

27. Ellis, A. W. M., and Swift, H. F.: Involvement of the Eighth Nerve in Syphilis of the Central Nervous System, J. A. M. A. **64**:1471 (May 1) 1915.

28. Leri, A.: Etude du nerf optique dans l'amaurose tabétique, Nouv. iconog. de la Salpêtrière **17**:358, 1904.

29. Schlagenhaufer: Anatomische Beiträge Zum Faserverlauf in den Sehnervenbahnen und Beitrag zur täbischen Sehnervenatrophie. Arbeiten aus dem Institut, für Anatomie und Physiologie des Centralnervensystems, No. 5, 1897; Abstr. Centralbl. f. Nervenh. u. Psychiat. **8**:564 (Neu Folge) 1897.

30. Stargardt: Ueber die Ursachen des Sehnervenschwundes bei der Tabes und der Progressive Paralyse, Arch. f. Psychiat. **51**:711, 1913.

process in which there is a proliferation of the endothelium and new formation of elastic membrane and increase in vessels.

3. Presence of *Abraumzellen* containing fat.

4. Increase in neuroglia.

5. Degenerative processes in axis cylinder and myelin sheath. No difference in time or extent of the degeneration could be made in the distal or proximal end of the orbital optic nerve and no conclusion, therefore, arrived at in determining an ascending or descending degeneration.

The changes in the retina are in general compared to those occasioned by pressure from a sclerotic internal carotid artery and also following experimental cutting of the optic nerve. The picture is different from that seen in ordinary toxic degenerations from quinin, felix-mas and atoxyl. Changes in the retina are found only when unmistakable changes in the nerve itself can be demonstrated; but with an intact retina early changes in the nerve may be shown. Schoenberg[31] accepts Stargardt's conclusions. In a comprehensive article dealing with the later investigations of the circulation of the cerebrospinal fluid and dye experiments, he supports the intraventricular or cranial subarachnoid route for treatment of optic nerve atrophy on the basis of a primary syphilitic process in the meninges.

### INFLAMMATORY MENINGEAL REACTIONS

We now come to the question of inflammatory meningeal reactions in tabes. Spiller's position has already been stated. Bresowsky[32] has found lymphocytic infiltration of the pia present in all of forty cases examined. This investigator working in Obersteiner's clinic, the seat of so much research on the pathology of tabes, believes that the thickening of the meninges is of distinctly inflammatory character and that its probable origin is from the blood vessels. The thickening pia may be observed in layers. This meningitis occurs undoubtedly in early tabes. The rôle of this meningeal inflammation in the pathogenesis of tabes is that of a toxic agent acting on the posterior roots in Obersteiner's area. He does not believe that pressure or sclerotic shrinking can explain the degeneration alone. Schroder[33] has found lymphocytic infiltration of the pia of inflammatory nature constantly present in five cases of tabes. This process was not limited to the pos-

31. Schoenberg, M. J.: Intracranial Treatment of Syphilitic and Parasyphilitic Nerve Affections, J. A. M. A. **66**:2054 (June 24) 1916.

32. Bresowsky, M.: Ueber die Veränderungen der Meningen bei Tabes und ihre pathogenetische Bedeutung, Arb. a. d. Neurol. Instit. Wien., **20**:1, 1912-1913.

33. Schroder, P.: Ein Beitrag zur Histopathologie der Tabes Dorsalis. Centralbl. f. Nervenh. u. Psychiat. **29**:585, 1906.

terior aspect of the cord but was also seen in the anterior septum. This investigator suggests that the diffuseness of the inflammation may explain the degenerations of the anterior and lateral columns when this occurs. Astwazaturow[34] reports a case of gastric crisis which he believes due to an underlying specific syphilitic meningitis involving the posterior spinal roots. His case did not show typical tabetic degenerations, and there was also evidence of a meningomyelitis present. The case might be regarded rather as one of pseudo-tabes. Schaffer, although an exponent of the toxic theory, states that in a number of cases of lumbar tabes he has observed also isolated degeneration of the cervical roots and corresponding degenerations in the bandelette externe. Starr (personal communication) relates a case in which this degeneration was found on one side of the cord only. Hassin,[35] in America, favors the meningeal theory, as does Williams.[36]

### ANOTHER GROUP OF CASES

There are a group of cases classified as pseudo-tabes syphilitica some with pronounced meningeal involvement. These types are aptly defined by Collins,[37] who has written on this subject as those in which clinically there is a picture of tabes and pathologically characteristic lesions of syphilis in the meninges or cord or both, but in which the distinctive lesions of tabes are not necessarily present.

Panegrossi[38] reports a case clinically diagnosed as tabes which revealed in the postmortem examination a striking accompanying pial meningitis in addition to the characteristic pathology of tabes (degeneration of the posterior root fibers in their extramedullary and intramedullary course, degeneration of the fibers in the Lissauer zone). The author raises the question whether in this case the cord changes may not be considered to be secondary to the spinal meningitis and refers to a number of reported cases in the literature referred to as cases of pseudo-tabes syphilitica: (Bibliography) Guiffre and Mitro, Lamy, Maplain, Fournier and Dieulafoy, Cardarelli, Garbini, Eisenlohr, Nonne, Oppenheim, Rump, Collins, Brash, Gaykiewiez and Schwartz.

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34. Astwazaturow, M.: Ueber Beziehungen zwichen Tabes und Leptomeningitis, Deutsch. med. Wchnschr. **37**:64, 1911.

35. Hassin, G. B.: Chicago Neurological Society, Meeting of Jan. 21, 1915, reported in J. Nerv. & Ment. Dis. **42**:699, 1915.

36. Williams, T.: The Pathogenesis of Tabes Dorsalis, Am. J. M. Sc. **136**: 206, 1908.

37. Collins, J.: Syphilitic Pseudotabes, etc., New York M. J. **77**:533, 581, 1903.

38. Panegrossi, G.: Beitrag zum Studium der vom chronischen spinal Meningitis begleitenten Tabes, Monatschr. f. Psychiat. u. Neurol, **23**:290, 1908.

On the basis of an anatomo-clinical study, however, in his case he has determined it to be one of cervical dorsal tabes combined with chronic spinal leptomeningitis.

Some mention should be made of combined tabes and amyotrophic tabes in considering the genesis of this affection. The underlying pathology of these complicating or associated conditions is as indefinite in the literature as is the case in the uncomplicated forms of tabes. The diffuse meningitis found by Schröder may be considered as a cause in this connection. Likewise, amyotrophic tabes may be explained by anterior root involvement and lower motor neuron disease. Wilson,[39] however, in a anatomo-clinical study of two cases is inclined to favor the toxic hypothesis in amyotrophic tabes, although there was a spinal meningitis present in both cases. It is, however, proper to here express the opinion of Derjerine in this regard. In discussing the rôle of meningitis in tabes he states: "Other factors besides meningitis are necessary to produce tabes. The best proof of this is that in individuals affected with syphilis paraplegia in consequence of meningo-myelitis, tabes is an exceedingnly rare complication."

## REPORTS OF SIXTEEN NECROPSIES

I have charted the necropsy records of sixteen cases of tabes dorsalis with this pathologic diagnosis. These cases were examined between the years 1898-1916 in the pathological department of the Stanford Medical School. Examination of the cerebrospinal fluid when made, the anatomic diagnosis, macroscopic and microscopic examinations of the spinal cord and meninges are noted. Abnormal changes in the meninges are printed in black face type. Thickening or cellular infiltration of the spinal meninges most marked in the posterior portion of the cord occurs in most cases; in fact, if we accept Case 1, in which no record of a microscopic examination is made, and Case 6, which is a classic description of combined sclerosis and not of tabes, one or the other of these processes is a constant finding. An instructive instance is that of Case 13 in which, with advanced atrophy in the posterior columns associated with marked fibrous thickening of the dorsal pia and adhesions between it and the dura the cerebrospinal fluid tests were entirely normal. No mention is made of a lymphocytic infiltration in this case, the assumption being that it did not exist, and the negative cerebrospinal fluid findings are thusly explained. Such a condition of the cerebrospinal fluid in cases of undoubted tabes has been reported among others by Purves Stewart,

39. Wilson, S. A. K.: The Pathology of Two Cases of Tabetic Amyotrophy, Rev. Neurol. and Psychiat. **9**:401, 1911.

TABES DORSALIS—SIXTEEN NECROPSIES

| Case No. | Pathologic No. | Initials, Age, Sex | Clinical Diagnosis | Necropsy Date | Examination of Cerebrospinal Fluid | Anatomic Diagnosis | Examination of Spinal Cord and Meninges | |
|---|---|---|---|---|---|---|---|---|
| | | | | | | | Macroscopic | Microscopic |
| 1 | I 34 | L. Y., male | No record | 9/24/98 | No record | Tabes dorsalis; atheroma; bronchopneumonia | Anemia of pia mater; posterior columns grayish white | No record |
| 2 | VI 33 | C. J., middle aged male | No record | 11/29/02 | No record | Tabes dorsalis; chronic spinal meningitis of posterior surface of cord with adhesions between dura and pia; degeneration of posterior white columns; aneurysm of ascending aorta with erosion of sternum, perforation into pericardium; hyperemia and edema of lungs; mucous bronchitis; hypertrophy of thyroid; chronic gastritis | Pia mater normal on anterior surface; pia mater on posterior surface; pia mater on posterior surface in lumbar and thoracic region thickened, adhesions between dura and pia; discoloration in column of Goll in upper dorsal and cervical regions on cut section | **Thickening and cellular infiltration on pia**; marked atrophy in median posterior white columns |
| 3 | VI 46 | Middle aged male | No record | 1/16/03 | No record | Tabes dorsalis; gray degeneration of posterior white columns of spinal cord; slight chronic meningitis spinalis and cerebralis; acute carbolic acid poisoning; edema glottidis | **Slight chronic meningitis**; gray degeneration of posterior white columns | No record |
| 4 | IX 70 | C. F., elderly male | No record | 3/ 9/06 | No record | Tabes dorsalis; chronic meningitis of cord, probably syphilitic; chronic cystitis; pyonephrosis; arteriosclerosis of aorta; bronchopneumonia | Pia normal on anterior surface of cord; on posterior surface hyperemic, edematous and thickened; adhesions between dura and pia throughout length of cord; no visible lesions on cross section | **Pia of posterior part of cord shows marked fibrous thickening with regular cellular infiltration**; slight degeneration of most peripheral fibers in posterior columns |
| 5 | X 23 | Male | Syphilis | 12/25/06 | No record | Tabes dorsalis; sclerosis of posterior white columns; meningoencephalitis and atrophy of cortex; mitral stenosis, hypertrophy of right side of heart; cyanotic atrophy of liver; bronchopneumonia; cystitis and pyelitis; atrophy of left kidney | Marked gray discoloration of posterior columns, no marked thickening of pia over sclerosis; hyperemia of pia with adhesions between dura and pia in cervical cord | **Moderate fibrous thickening of pia and some cellular infiltration**; very many amylaceous bodies in diseased parts of cord; marked sclerosis of posterior white columns |
| 6 | XI 30 | S. A., 51, female | No record | 2/ 7/08 | No record | Tabes dorsalis; recent degeneration of motor tract; embolism, main right branch of pulmonary artery; fatty heart; thrombosis of both femoral veins; brown atrophy of heart and liver; chronic gastritis; adhesions in left half of abdominal cavity and pleura | Spinal canal and cord macroscopically normal | No meningitis; degeneration of the median portion of posterior white columns, also of lateral columns; degeneration of crossed and uncrossed pyramidal tracts with vacuolation |
| 7 | XV 63 | R. R., 60, male | Locomotor ataxia, perforating ulcer | 1/23/12 | No record | Tabes dorsalis; perforating ulcer of feet; chronic suppurative cystitis and pyelitis; emphysema; adhesive pleurisy; fibrous pericarditis; arteriosclerotic kidneys; parenchymatous degeneration of liver and kidneys | **Grayish thickening of pia on posterior surface of cord**; cut section of cord shows grayish discoloration of median columns in cervical region and beneath thickened pia in lower dorsal region | **Marked fibrous thickening of pia on all sides of cord**; marked degeneration of posterior white columns |
| 8 | XV 74 | K. W. F., 54, male | Chronic gastritis, terminal pneumonia | 2/24/12 | No record | Tabes dorsalis; chronic posterior meningomyelitis; sarcoma of dura mater; purulent cystitis, chronic; pyonephrosis; emphysema scars at apices, bronchopneumonia; recent fibrous pleurisy; sarcoma of dura mater of brain, left side | Pia mater shows a whitish thickening and marked edema on posterior surface of cord; cut section of cord, posterior white columns distinctly gray | **Marked fibrous thickening of pia over posterior white columns**; these show well marked degeneration at all levels |

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|---|---|---|---|---|---|---|---|---|
| 9 | XVI 136 | F. P., 59, male | Tabes dorsalis | 1/ 6/13 | No record | Tabes dorsalis; atheroma of aorta severe, probably syphilitic; arteriosclerotic scars in right kidney; pseudomembranous cystitis; prostatic abscesses; pyelonephritis; adhesions in both pleural cavities; hypostatic pneumonia | Pia mater over posterior surface of cord shows a white thickening from lower end to certical region; cut section of cord shows a very marked gray discoloration of posterior columns throughout cord | **Thickened and infiltrated meninges around whole of cord most marked in posterior and lateral aspects**; marked degeneration of posterior columns |
| 10 | XVII 45 | R. L., 37, female | Tabes (?), tuberculosis of lungs and left hip, tuberculous meningitis | 11/14/13 | Five leukocytes per c.mm. | Tabes dorsalis; tuberculosis of lungs (chronic), kidneys, intestines and left hip joint; chronic salpingitis; colitis acute | Pia mater on anterior surface of cord normal; posterior surface shows whitish thickening, and some hyperemia over lower portion of cord; marked degenerations of posterior columns throughout cord | **Some cellular infiltration and moderate fibrous thickening of pia especially on posterior aspect of cord**; degeneration of nerve fibers most marked in Goll's columns |
| 11 | XVII 169 | D. M., 49, male | Tabes doraslis | 5/25/14 | No record | Tabes dorsalis; bronchiectasis chronic; bronchopneumonia; pyelitis; cystitis; abscess of kidney multiple | Cut sections of cord show distinct degenerations in posterior columns, most striking in cervical region, also degeneration in Clarke's columns | **Thickened pia with cellular infiltration over dorsal surface of cord** most marked in dorsal and lumbar region; degeneration of posterior columns of cord |
| 12 | XVIII 44 | L. O., 44, female | Tabes dorsalis, endocarditis, cystitis, pyelitis | 10/10/14 | Globulin increased, leukocytes 15.4 per c.mm., Wassermann +++ | Tabes dorsalis; pelvic peritonitis acute; cystitis acute; pyelitis acute; endocarditis chronic; bronchitis acute; bronchopneumonia | Pia mater over anterior surface of cord normal; over posterior surface there is an edematous thickening and hyperemia; cross section of cord shows a slight grayish discoloration of posterior columns in lumbar region | **Marked fibrous thickening of pia with lymphocytic infiltration**; extensive degeneration in posterior white columns |
| 13 | XVIII 80 | H. P., 56, male | No record | 12/14/14 | Wassermann negative, cell count and globulin not increased | Tabes dorsalis; pyelitis chronic; pyelonephritis; dysentery (?) | On dorsal surface of cord many adhesions between dura and pia; cut sections of cord show grayish discoloration of posterior columns especially in cervical region | **Marked fibrous thickening of pia over posterior surface of cord**; very marked atrophy in posterior white columns |
| 14 | XVIII 117 | W. W. T., 45, female | Tabes dorsalis, arsenical poisoning | 1/28/15 | Wassermann negative, globulin positive, leukocytes 11.2 per c.mm. (Wassermann negative blood) | Tabes dorsalis; cystitis; pyelitis; pyelonephritis acute; bladder perforation of; peritonitis acute; arsenical poisoning acute (salvarsan); colitis ulcerative | **Slight adhesions between dura and cord on posterior surface in cervical region; pia mater is slightly injected but not definitely thickened**; no visible degeneration on cut surface | In lumbar region marked degeneration in posterior white columns, extending across from one side to the other between central canal and half way to posterior surface; slight but definite lymphocytic infiltration of spinal pia |
| 15 | XIX 86 | M. J. L., 54, male | Tabes dorsalis | 1/23/16 | No record | Tabes dorsalis; cystitis chronic; pyelonephritis acute; ulcer of foot, perforating; syphilis of aorta; abscess of heart muscle, metastatic | Dorsal pia of cord thickened, opaque and hyperemic along entire surface; a lardaceous and hyperplastic meningitis is seen in dorsal cord; ventral aspect of cord does not appear to be involved; cut section of cord shows extensive degeneration of posterior columns | **Marked fibrous thickening of pia especially on posterior aspect of cord**; atrophy of nerve fibers in posterior white columns |
| 16 | XIX 141 | O. L., 50, male | Tabes dorsalis | 4/15/16 | Wassermann positive, globulin positive, leukocytes 39 per c.mm. | Tabes dorsalis; cystitis acute; bronchopneumonia | Pia over dorsal surface of cord somewhat thickened; on cut section the dorsal columns are distinctly yellowish brown | **Marked fibrous thickening of pia surrounding cord, most marked on posterior and lateral surfaces**; extensive degeneration of posterior white columns, also degenerations in one crossed pyramidal tract and in the uncrossed pyramidal tract of the opposite side |

Babinski, Ferrier and by Nonne. In the Stanford Clinic such a case is occasionally met with. But we have elsewhere maintained and still hold that a progressive tabes will at one time or another time show a cerebrospinal fluid reaction characteristic of subacute inflammatory meningitis. Andre Leri[40] has reported a case of meningomyelitis in which the meninges at different levels of the cord presented different pictures: at one level definite thickening of meninges without cellular infiltration, and at another level meningeal thickening plus cellular infiltration. Case 11 (colored insert) is interesting in this regard, a section in the lower dorsal region showing a condition in the meninges somewhat similar to the foregoing. Over an area of previously thickened membranes there is a superimposed cellular infiltration suggesting the progression of the meningeal lesions in different stages: the first stage of inflammation having subsided, there resulted a fibrous thickening, and after a certain remission a recurrence of the meningeal infiltration with characteristic fluid changes. (No record of these fluid changes in the charted case.) In Case 14 (see figure) a section at $L_1$ shows the degeneration described by Pierret.

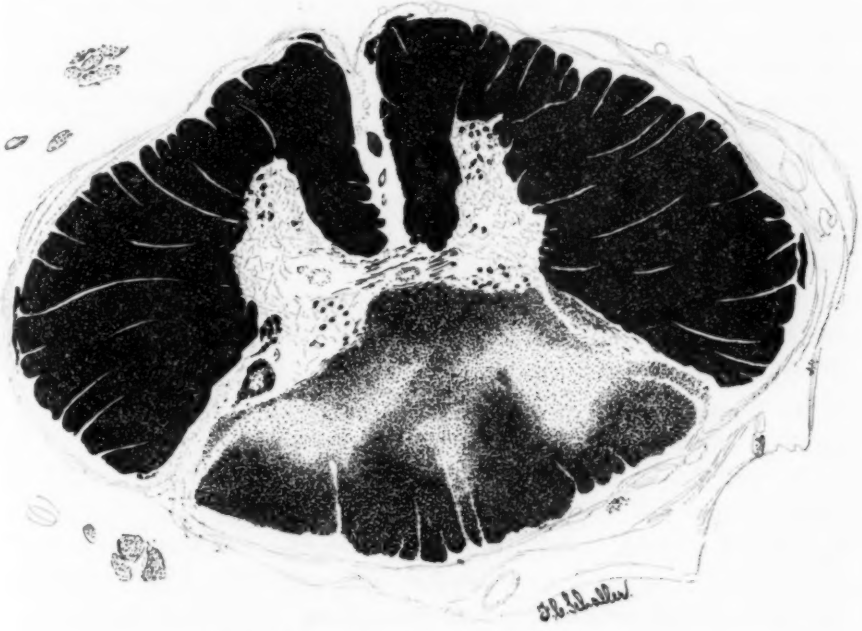
An examination of the table will show the distribution and extent of the meningeal changes. It is noteworthy that these findings were for the most part established in a general pathologic laboratory, and were noted not as a particular study of tabes, but as part of the general pathologic survey of the case. It is possible, as has been maintained by Bresowsky, that by improved staining methods, cellular infiltrations including the presence of plasma cells might be demonstrated in practically all of these cases.

Spielmeyer,[41] in a recent review of articles dealing with the pathologic anatomy of tabes states that the advocates of the meningeal theory are less often heard from now than formerly. He denies that an inflammatory meningeal reaction is a constant finding in tabes. We are not able to follow Spielmeyer in all his arguments. Spielmeyer's trypanosome dogs died of a subacute general infection. Microscopic examination of the spinal cord showed some slight degeneration of the cerebellar tracts in addition to the degeneration in the posterior columns, and our impression from reading Spielmeyer's article was that even in the posterior columns the degeneration was slight. No cerebrospinal fluid examination was made, and although no meningeal

40. Leri, A.: Soc. Neurol., Paris Séance, Dec. 30, 1911, Rep. Rev. neurol. **22**:785, 1911.

41. Spielmeyer, W.: Paralyse Tabes Schlafkrankheit, Ergeb. der Neurol. u. Psychiat. **1**:217, 1912.

inflammation was found it is not stated that this examination included an examination of the radicular nerve (Nageotte). Even in the presence of optic atrophy and fifth nerve involvement a primary toxic combined sclerosis must be considered here. It is to be regretted that the trypanosome strain which Spielmeyer used in these experiments had died out, and that we have not been able to find a record of a repetition of these experiments. In stovain tabes his finding that a direct neurotrophic action of the drug acts on the extramedullary portion of the posterior root fibers would not appear to support the theory that a syphilitic toxin would act differently, that is, on the intramedullary portion of these fibers and spare other intramedullary fiber systems.



Bandelette of Pierret. $L_1$—Weigert stain.

The tabulated list presented in this study shows the constancy of meningeal changes in tabes, as has been noted particularly of late in the special investigations above mentioned (Spiller, Bresowsky, Schroder). Assuming that the cerebrospinal fluid reactions are due to the presence of a subacute syphilitic meningitis we would emphasize the importance of fluid tests in the routine examination of a case of tabes, and would even go so far as to say that the information obtained from a characteristic positive fluid in a progressive tabes is one of the greatest aids to diagnosis that we possess in this affection. Such

being the case the pathologic condition which it evidences in the meninges would give this condition the dignity of a basic pathologic condition rather than a complication which the supporters of the toxic and other theories would have us believe is the case.

Not alone is the pial and arachnoid meningitis a *basic* pathologic condition, but it is in all probability a *primary* one in the affection we are considering. Since Ravaut, in 1903, called attention to the involvement of the nervous system (alterations in the cerebrospinal fluid) without nervous symptoms in cases of general infection of syphilis his findings have been corroborated by a number of subsequent investigators. Nichols,[42] for example, has dwelt on the important fact that the cerebrospinal meninges may be invaded in the primary and secondary stages, and he has elsewhere expressed the opinion that this local infection is not temporary and comparable to the skin rash, but persists to later be associated with those pathologic conditions which underlie certain definite types of nervous syphilis, as for example, tabes. Quite recently Fildes, Parnell and Maitland[43] studied a large number (624) of unselected cases of syphilis and found pleocytosis in 18 per cent of these. The occurrence of a positive Wassermann reaction and the demonstration of the *Spirocheta pallida* established the syphilitic nature of the process. In 80 per cent. of these cases which presented abnormal fluids, there were no clinical symptoms or signs of nervous disease.

### MENINGEAL THEORY

In support of the meningeal theory of tabes we may mention the findings of Mott in dourine, of Long in pellagra, and especially the recent pathologic studies of optic nerve degenerations in tabes which appear almost conclusive. We have been accustomed to typify a toxic degeneration of the spinal cord by the subacute combined degenerations observed so often in association with severe anemias. But here surely the picture is quite different, there being no inflammation present and the degeneration is not limited to one system. The degenerations found by Rothmann and by Schroder in monkeys may belong to this type.

The ergot degeneration found by Tuczek appears to be similar to that observed in tabes, but in the absence of modern staining methods we are not prepared to accept the pathologic findings as conclusive.

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42. Nichols, H. J.: Observations on the Pathology of Syphilis, J. A. M. A. **63**:466 (Aug. 8) 1914.

43. Fildes, P., Parnell, R. J. B., and Maitland, H. B.: Unsuspected Involvement of the Central Nervous System in Syphilis, Arch. Neurol. and Psychiat. **1**:231, 1919.

The functional behavior of the cerebrospinal fluid in which the meninges are suspected of being originally affected has been the subject of a series of studies in the Stanford Clinic. Mehrtens and West[44] have studied the absorption of the cerebrospinal fluid in tabes among other conditions by means of phenolsulphonepthalein injected into the spinal subarachnoid space and later recovered from the urine. Under normal conditions the appearance time is within ten minutes of injection, but in disease of the central nervous system, especially when the meninges are involved, this time is appreciably lengthened. In nine cases of tabes presenting both positive clinical and positive fluid tests the appearance time of the dye was thirty minutes or over, with but one exception. In five additional cases of clinical tabes with negative fluid findings throughout, including the Wassermann reaction and gold sol test, the average excretion time was forty minutes. Of these five cases, three had negative fluids when first observed, and two became negative following treatment. The importance of this test as a means of diagnosis in such cases is apparent. In the above study no conclusion was arrived at as to the location of the absorbing tissues. Recently Dandy and Blackfan,[45] and Cushing and Weed[46] have studied the question of fluid absorption. The former investigators believe that the absorption of the cerebrospinal fluid takes place as a diffuse process within the whole subarachnoid space directly into the blood by means of the capillaries. A double ligation of the spinal dura in the dorsal region prevented fluid circulation, and injection distal to the ligation showed reappearance of the dye in the blood. They reject the contention of Mott and of Goldmann that the lymphatics play any appreciable part in this absorption process. Cushing and Weed believe that the most important channels of absorption are by means of the arachnoid villi into the large cranial venous sinuses. Damaged meninges with impaired function in tabes might thusly explain delayed absorpton time.

In a paper shortly to be published, Mehrtens, in the Stanford Clinic, has extended his studies of the cerebrospinal fluid to a consideration of cerebrospinal fluid pressure in those diseases in which as a result of chronic inflammatory processes the meninges show destructive changes. Not only is fluid pressure increased in cases

44. Mehrtens, H. G., and West, H. F.: Absorption of Phenolsulphonephthalein from the Subarachnoid Space in Diseases of the Central Nervous System, Arch. Int. Med. **20**:575, 1917.

45. Dandy, W. E., and Blackfan, K. D.: Internal Hydrocephalus, Am. J. Dis. Child. **8**:406, 1914.

46. Cushing and Weed: Studies on the Cerebrospinal Fluid, J. M. Res. **31**:1, 1914.

which show positive laboratory evidence of inflammation, but it persists in cases of tabes and cerebrospinal syphilis in which the fluid reaction has become normal following specific treatment, or was normal when first investigated, as is possible in certain stages of tabes. In this latter group of negative fluids in tabes an increased pressure of fluid from 150 to 250 mm. of water has been found in 85 per cent. of the cases. This test points to damaged meninges, and is therefore of diagnostic importance.

From the foregoing I am an adherent to the meningeal theory of tabes in the sense that the meningeal inflammation occasions in some manner the characteristic degenerations of the posterior roots and the posterior columns of the cord. The exact manner in which this takes place is still uncertain. Although the necropsy study was limited to the cord meninges, I do not wish to assert that this is the only location of such a process, for the frequency of Nageotte's meningomyelitis has been proved by a number of different investigators. I believe that the slight thickening of the meninges noted by the earlier writers is always inflammatory in origin. And although in comparison with other disease of apparently more severe meningeal lesions with intact root fibers, we must not lose sight of the fact that tabes runs an exceedingly chronic course, punctuated with remissions and exacerbations. I have reported a case of tabes first in which the incubation period was forty-four years. How, then, may these meningeal lesions act? We may mention the following possibilities:

1. *Mechanically:* (*a*) By fibrous construction of the dorsal roots in their perforation of the spinal pia at Obersteiner's area. (*b*) By root pressure from increased cerebrospinal fluid pressure in this same locality.

2. *Inflammatory:* By direct extension of meningeal inflammation causing a meningo-radiculitis as in Nageotte's area.

3. *Toxic:* By toxic products engendered by the meningeal inflammation in the subarachnoid space, and affecting the dorsal roots.

## THERAPEUTIC INDICATIONS

In the Stanford Clinic we have based certain indications of therapy in tabes on the above conception of a primary inflammatory meningitis as being the principal pathogenetic factor in tabes. So long as increased cell count and protein content exist in the fluid the process is active and must be treated. If the ordinary intravenous arsphenamin and ordinary mercurial treatments are powerless to modify these abnormal states then the intraspinal methods of therapy are indicated. Exceptionally, when special cranial complications arise, intracranial methods may be likewise indicated. In not a small

experience, extending now over a period of a number of years, we believe that the fluid may be cleared in tabes by intraspinal therapy, when previous intensive intravenous treatment fails to produce this result. The Wassermann reaction, increased pressure of the fluid and delayed absorption time are more difficult to modify, not being dependent of active meningeal processes. In the case of a negative fluid indicating an arrest of meningeal inflammation we believe that intraspinal treatment is contraindicated.

## CONCLUSIONS

1. Tabes is due to a degeneration of the posterior spinal roots.

2. Subacute syphilitic inflammatory changes in the subarachnoid space (posterior leptomeningitis, meningeal and neural involvement of radicular nerve) are in etiologic relationship with the degeneration of the posterior roots. Likewise similar processes explain the cranial nerve involvement in tabes.

3. The manner in which this subacute inflammatory meningitis produces root degeneration is as yet uncertain: it may act by direct extension of the meningeal lesions to the nerve roots causing a meningoradiculits; or by pressure constriction from sclerosed meninges; by toxic products engendered by this inflammation; or even by increase of fluid pressure as is the case in posterior spinal root degeneration in brain tumor. These causes may act together or independently.

4. This inflammation, which is constant in tabes to a greater or lesser degree, is evidenced by characteristic cerebrospinal fluid changes at one time or another in every case of tabes.

5. Specific therapy in tabes should have for its object the reappearance of normal fluid reactions. Intraspinal therapy may be necessary to attain this object.

6. Conversely intraspinal treatment is contraindicated when the fluid shows no inflammatory reactions.

# SOME ADVANCES IN THE SURGICAL TREATMENT OF INJURIES OF THE NERVOUS SYSTEM

A CRITICAL REVIEW

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"He who desires to learn the practice of surgery must go to war." Thus spoke the "father" of medicine more than 2,000 years ago, and his statement is, in a sense, applicable to the surgical experiences of the world struggle that has just ended. The war was fought not by armies but by nations, and every element in the communities was mobilized for war purposes. The medical and surgical treatment of the enormous number of wounded was a problem not only for the military surgeon in the front, base and home hospitals, but also for the scientific laboratory investigator at home.

To give, therefore, a brief account of the advances during the war, in the surgical treatment of injuries of the nervous system, demands a consideration not only of the actual improvements in operative methods, but also of the laboratory and clinical investigations of war problems which have made these surgical advances possible.

The following summary deals only with the real progress that has been made and is therefore not a review of everything that has been written on the subject. No consideration will be given to details of surgical technic which have no interest for the neurologist, and no references have been made to publications in which no new and useful methods have been described. A large part of this review is a summary of facts derived from personal communications made to the writer together with a statement of his own experiences in the surgical treatment of war injuries of the central and peripheral nervous systems in home hospitals.

## I. INJURIES TO THE SKULL, WITH REFERENCE TO THE CRANIAL CONTENTS

The problems of intracranial surgery for injuries of the brain have been mainly those that have to do with penetrating wounds of the skull, and the surgical treatment of these wounds has been in the main a question of prevention of infection and meningitis. Fractures of the skull, without injury to the scalp or dura, with which were associated intracranial lesions, did not present any problems which had not been met with in the surgery of civil life, and no new indications or methods of treatment have been evolved.

In the beginning of the war it was the common practice of military surgeons to open wounds of the skull widely and to drain them extensively. By free drainage of the wound infection was to be prevented. Very soon, however, the method of treatment was found to be unsatisfactory. Free drainage did not, in many instances, prevent infection; secondary suppuration occurred and large brain hernias, with all their dangers, resulted. The attempt was then made to apply the principles of "debridement" to skull and brain wounds, as this method of treatment had given such good results in wounds of other parts of the body.

The viewpoint which led to this change in the surgical methods was the following: In every patient who has sustained a wound of the skull and brain, a certain amount of infectious material has been implanted in the wound. If the patient is seen early enough (within from twenty-four to forty-eight hours) the removal of the tissues into which infectious materal has been implanted should remove the danger of infection. If infection has already occurred, a wide extirpation ("debridement") of the infected tissue might still prevent the spread of the infection. In either case the surgeon should, as far as possible, operate in healthy tissues. This was possible as far as scalp, bone or dura were concerned. Obviously, however, wide excision of contused or pulpified brain tissue was impossible, and all that could be done was to remove, by irrigation and other mechanical means, whatever tissue was softened and destroyed. The best results were obtained by the method of "excision en bloc." The wound in the scalp was excised, the operator keeping in healthy tissue well away from the injured area. In a similar way a piece of bone was removed in one piece and an area of dura around the point of injury was excised. The softened brain tissue was then carefully washed away by a stream of saline solution. Cushing[1] inserted into the track in the brain a flexible soft rubber catheter which was used both for palpation and search for foreign bodies and for suction purposes. By attaching to the end of the catheter a Carrel-Gentile glass syringe with its rubber bulb, softened brain tissue, fragments of bone and small foreign bodies could be sucked up into the catheter. By gentle irrigation through the catheter additional destroyed tissue and bone fragments could be removed. Larger foreign bodies, if they lay in the track, could be palpated and removed with delicate forceps. Only those that are easily accessible should be extracted; those whose removal would surely increase the damage already done, should be allowed to remain. Other surgeons, however, did not use the catheter method, but

1. Cushing, Harvey: Notes on Penetrating Wounds of the Brain 1:221 (Feb. 23) 1918.

mechanically removed the cerebral debris.[2] Cushing injected into the track a small quantity of dichloramin-T in eucalyptus oil. The dura was then closed by suture, a plastic operation, or by fascial transplantation, and the wound in the scalp closed without drainage—also by a plastic operation if necessary.

From the time that this technic was introduced (De Martel, Cushing, Gray) there was a steady fall in the mortality of cranial operations, meningitis became more rare and hernia of the brain unusual. Cushing states that before this method of treatment was introduced, at least 70 per cent. of the patients operated on succumbed at the forward or base hospitals, and his contention is supported by the early statistics.[3] On the other hand, later statistics show a mortality of only 20 to 30 per cent. (Cushing, 28.8 per cent.).

The majority of the wounds which could be treated by the method above outlined, healed by primary union. When this "excision en bloc" was thoroughly carried out, it was found that, if necessary, an uninjured dura could be incised with comparative safety. Extensive shell wounds with great loss of substance, however, had still to be treated by the open method, and in these the mortality from infection and meningitis remained high. The majority of these cases, however, were, on account of the extensive injury to the brain, hopeless from the very beginning.

To a very great extent in the operations in the hospitals of the American Expeditionary Forces, and to some extent in the hospitals of the English and French, local anesthesia was substituted for general anesthesia in many cranial operations. The most extensive cranial operations were performed under local anesthesia with novocain. The violent respiratory efforts made by a patient who is being anesthetized by ether are accompanied by sudden increases and decreases of intracranial pressure, and these are very apt to diffuse disorganized and softened brain tissue over the meninges, and thus spread infectious material. Cushing claims that the use of local anesthesia in operations for the cranio-cerebral wounds of warfare, has distinctly contributed to improved results.[4]

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2. De Martel: In Chatelon and De Martel, Blessures du Crâne et du Cerveau, Masson et Cie, 1917; Gray: Brit. M. J., 1916; Williams and Albert: Bull. et Mém. de la Soc. de Chir. de Paris **44**:7 and 36, 1918.

3. Abadie: Presse méd., 1916, 56 per cent.; Cuneo: Presse Méd., 1916, 60 per cent.; Sencert: Lyon Chir., 1916, 63.5 per cent.; Moulonguet et Legrain: Presse Méd., 1918, 62 per cent.

4. Cushing: Loc. cit.; De Martel: Bull. et mém. Soc. de Chir. de Paris, **44**:1364, 1918.

Perhaps the most important laboratory work in connection with the subject of meningitis, is that of Weed,[5] in which he showed that, in animals who had received intravenously an otherwise non-toxic dose of bacteria, the withdrawal of cerebrospinal fluid by lumbar puncture was very apt to be followed by meningitis on account of changes in the permeability of the choroid plexuses. While there is, as yet, no evidence that the results obtained by animal experiments are applicable to man, the evidenee is sufficient to make lumbar puncture in threatened meningitis or in patients who have sustained a cerebral injury and in whom a meningeal infection is possible, inadvisable unless very specially indicated.

The experiences of the Medical Corps of the American Army are too recent to permit one to say much regarding the treatment of the secondary and late complications of brain wounds. The best résumé of the subject from other sources is that of Gordon Holmes,[6] based on the examination of 2,357 patients, of whom 526 were personally observed.

Secondary abscesses of the brain are most apt to occur between three and six months after the wound, but they may occur at any time in patients with an unhealed wound or a cerebral hernia. They may occur under a necrosed fragment of the skull, or deep in the brain around a fragment of indriven bone or a foreign body, or at some distant region of the brain not affected by the primary wound. As is well known from experiences in civil life, these abscesses are very apt to occur after wounds or infections in the orbit or ethmoid sinuses. Of 37 cases that were operated on, 28 terminated fatally. The best treatment consisted in puncture or evacuation through a small incision and drainage. Gordon Holmes states that the results were not satisfactory. (In the few cases operated on personally by the reviewer, the results have been excellent.) It is my opinion that the percentage of good results will vary with the surgical method adopted and especially with the care in the after-treatment of the patients. It is poor practice, after a brain abscess has been drained at an operation, to remove the drainage tube at each subsequent dressing and to reinsert it. After a drainage tube is once in place and is fixed to the scalp by suture so that it cannot be disturbed, it should never be removed as long as there is any drainage at all. When the amount of drainage has become small, the tube should be shortened, but it should never he removed and replaced during the entire surgical treatment.

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5. Weed, Wegeforth, Ayer and Felton: The Production of Meningitis by the Release of Cerebrospinal Fluid, J. A. M. A. **72**:190 (Jan. 18) 1919.

6. Comptes Rendus de la Conférence Chirurgicale interalliée pour l'étude des plaies de guerre, Novembre, 1917.

This I believe to be the "enacting clause" in the successful treatment of late brain abscesses.

As we have already stated, the frequency of cerebral hernia was much diminished with the improved treatment of the cranio-cerebral wounds If a cerebral hernia persists for a very long period or develops late, an infection of the brain tissue is present in the large majority of cases. The attempt to resect the hernias was followed, as was to be expected, by disastrous results, and this method of treatment was soon discontinued. The statement that a hernia which persists more than three months or one that appears after this period of time usually means that there is an infection within the brain tissue—most often an abscess—is justified. The treatment of these late abscesses in brain hernias should not differ essentially from the treatment of late abscesses without cerebral protrusion.

We cannot leave the subject of the treatment of cranio-cerebral wounds without mentioning the fact that many excellent studies of the symptoms of brain lesions have been published, which have contributed to clarify certain clinical pictures and have hence contributed distinctly to improving the indications for operation in some intracranial injuries. Thus mention must be made of the so-called "longitudinal sinus" syndrome, that peculiar bilateral spasticity which results from compression of the longitudinal sinus by depressed bone. Then also the fact, mentioned by very many writers, that surprising improvement may occur without operative interference in many apparently extensive and hopeless cerebral injuries.

From the standpoint of fatality, it soon became evident that injuries of the transverse and lateral sinuses were far more dangerous, and the mortality with or without surgical interference far greater in these than in injuries of the longitudinal sinus. This was well shown, for example, by the statistics of Velter;[7] all but one of the patients with a laceration of the transverse sinus died, usually from hemorrhage; the majority of those with an injury of the longitudinal sinus — unless the injury of the brain was excessive — recovered.

Lack of space prevents the mention of many excellent clinical papers.

### II. INJURIES TO THE SPINAL CORD

The subject of the surgical treatment of wounds of the spinal cord can be, unfortunately, dismissed with a few words. As was the case in the Crimean campaign, in our own Civil War, and in the Boer War, the injuries of the spinal cord by bullets or other missiles have been to a great extent, irremediable. As a group, spinal cord injuries

7. Velter: Les Plaies du Crâne, 1917.

are the most deplorable of all war injuries. While, rarely, a bullet or shrapnel ball may enter the spinal canal without injury of the cord, a complete division or transverse softening is usual. The experiences of the war have shown that the spinal dura should never be opened if it is intact, and in these cases the wound of the soft parts and the bone should be treated by "debridement" and the Carrel-Dakin method of irrigation. From the technical surgical standpoint, no advances have been made in spinal surgery as a result of the experiences of the war. On the neurological side, however, many valuable facts have been recorded. As some of these have a bearing on the indications for treatment and on prognosis, they are deserving of mention.

The reports of Head and Riddoch[8] have once for all given the coup de grace to the so-called Bastian-Bruns law, and have definitely proved that spasticity and exaggeration of reflexes can and do frequently occur after a complete transverse cord lesion. Some of the cases reported by these authors had a large defect in their cord, as proven by operation or necropsy. Therefore, spasticity is not of necessity a sign of an incomplete cord lesion in patients seen a few months or longer after their injury. The same authors have contributed a paper to the same journal,[9] in which, in a study of automatic bladder activity after complete spinal lesions, they give very useful suggestions as to the treatment of the bladder symptoms in spinal cord lesions.

The experiences of the war have shown that spinal concussion is very frequent, mainly because of the extensive use of high explosives. It had already been known, of course, that spinal concussion could occur from the impact of a bullet on part of a vertebra, and that the symptoms of concussion varied from slight ones to those of a complete transverse cord lesion. What we have learned from this war is that such concussion often occurs without external injury of any kind when a high explosive shell bursts in close proximity to the individual. The symptoms, due to small hemorrhages into the spinal cord or to air emboli, may cause secondarily a transverse softening of the cord with a permanent transverse lesion. On the other hand, great improvement, and sometimes even complete recovery, may occur within a few months of the injury. (We have personally seen several soldiers who were invalided back to the United States with supposed complete transverse lesions of the spinal cord from concussion, who began to improve soon after their return, and who have, to a great extent, recovered.)

8. Head and Riddoch: Brain, Lond. **40**:264, 1918.
9. Head and Riddoch: Ibid. **40**:188, 1918.

Roussy and L'Hermite[10] have given an excellent description of these cases of spinal concussion. These authors have also paid much attention to the symptoms of complete and incomplete spinal lesions. They state that in some cases with apparently the symptoms and signs of a complete transverse cord lesion, careful examination will reveal that some sensation persists in the perineum and over the coccyx. (We have carefully looked for this in a number of patients, but as yet, have failed to find it in any cases of so-called complete lesions.)

### III. INJURIES OF THE PERIPHERAL NERVES

There never has been before and perhaps there never again will be an occasion like the present for the study of the anatomy and physiology of the peripheral nerves, the symptoms of complete and incomplete nerve lesions, the manner and the course of nerve regeneration, the technic of neurolysis, nerve suture, nerve transplantation, etc., and the results that can be obtained by surgical treatment. The literature of the subject is already very large, and is still growing. The importance of the subject is universally recognized, and careful studies are being made in all countries. The Surgeon-General of our Army has appointed a special commission of neurologists, surgeons and neuropathologists to report on the subject of peripheral nerve injuries. A large amount of useful information has already been collected. It is certain that, as a result of the experiences of the great war, a thorough and profound knowledge of peripheral nerve injuries will have been gained. Already excellent textbooks on wounds of the nerves (Tinel, Athanassio-Benisty, etc.) have been published.

Within the compass of this short review only a few points can be touched on.

A good knowledge of the finer anatomy of the nerves and the exact levels at which sensory and motor branches leave the main trunk, is important for the neuro-surgeon and for the neurologist, and the experiences of the war have given us a very large amount of information regarding the normal and the many variations from the normal that may be encountered.[11]

Much information has also been gathered regarding nerve pattern and the course of fibers in the peripheral nerves. This was a subject to which very little attention had been paid in the past — a subject of

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10. Roussy and L'Hermite: Blessures de la Moelle et de la Queue de Cheval, Masson et Cie, 1918.

11. Tinel, loc. cit.; Bonisty, loc, cit.; Borchard and Wjasmenski: Bruns' Beitr. **107**:353, 1917; Verga: Compt. rend. de la Conférence Chirurgicale interalliée; Burrows, Brit. M. J., p. 1199, 1918. Rauschburg: Bruns' Beitr., 1918; etc.

practical importance for the surgeon.[12] Thus, for example, motor fibers to the pronator radii teres run down on the outer side of the inner head of the median and then along the outer aspect of the main trunk of the nerve, while the fibers to the flexor carpi radialis run down the outer side of the outer head and then run down the trunk of the nerve with the fibers to the pronator teres. The fibers to the flexor sublimis digitorum run down on the posterior and inner aspects of the inner head and the trunk of the median nerve together with the fibers to the flexor profundus digitorum, flexor longus pollicis and pronator quadratus. The sensory fibers occupy the most central portion of the median trunk. The course of sensory and motor fibers has been traced in all of the large nerve trunks, and it is clear that this knowledge is very important for those who have to make indications for operative interference in incomplete nerve injuries.

We cannot enter on the subject of the symptomatology of nerve lesions except to state that we now possess a good knowledge of the typical symptoms of complete and incomplete nerve lesions and of the many variations from the typical pictures that are met with. Careful observations have proved that there is never complete recovery of tactile sensation nor of active motor power for at least three months after the suture of a divided nerve. According to Gosset, the average time before the return of the first voluntary movements is six months for the median, eight months for the musculospiral, ten months for the external and eleven for the internal popliteal, eleven months for the ulnar, and sixteen months for the sciatic nerves. Joyce[13] has seen improvement within three months and complete recoveries within fifteen months. Several years will have to elapse, however, before very large statistics on end-results will be available. There is not any doubt that the earlier a divided nerve is sutured, the more rapid and the more complete will be the nerve regeneration. The large majority of the wounds in the war were complicated by infection and therefore there was a necessary delay before nerve suture could be performed. It has been the custom of most operators to wait at least three months after a wound that had been infected was completely healed before a nerve suture was undertaken, in order to avoid a possible infection of the newly made wound and nerve suture from a dormant focus.

Careful and repeated neurological examinations must precede every nerve operation, because only by this means is it possible to determine whether the nerve in question is completely or incompletely interrupted and whether any regeneration has occurred. If the interruption is not a complete one, then the surgeon must be conservative in his

12. Stoffel; Marie, Meige et Gosset: Compt. rend. Acad. de méd., Dec. 28, 1915; Déjirine, Presse méd., No. 13, 1916; etc.

13. Joice, J. L.: Brit. J. Surg. **6**:418 (Jan.) 1919.

surgical methods, or if some regeneration has occurred, conservative surgery is also called for. In this type of case, neurolysis—the freeing of the nerve from compression by scar tissue—and perhaps partial resection of a nerve is all that is required. Neurolysis will often suffice. If the examinations showed that part of the nerve was completely interrupted and had not improved (and to arrive at such a conclusion a knowledge of nerve pattern is indispensable), the operator may find a bulbous thickening of only that part of the nerve. In such a case, resection of the bulb (part of the thickness of the nerve trunk) may be necessary. Experience has shown, however, that the surgeon should always stimulate all sides of the nerve trunk electrically before determining to divide part of a motor or mixed nerve.

The same statement must be made for all nerve resections. As long as the gross continuity of the nerve remains, even if there is a large bulb, this bulb should never be resected before the exposed nerve has been tested with the electric current.

If a nerve bulb is to be resected, the surgeon must remove successive transverse sections of the bulb until distinct funiculi can be seen in the transverse section.

End to end suture should always be accomplished without tension, and can often be successfully done if the extremity is placed in the most favorable position for relaxation of tension. Very fine silk on very fine cambric needles should be used, and the nerve ends should be placed in as near the normal relation to each other (without rotation) as possible. Professor Huber, of Ann Arbor, who has done an enormous amount of experimental and pathologic work on nerve suture and nerve transplantation, states that these fine sutures can be passed through the entire thickness of the nerve trunk.

If, after resection of a bulb, the nerve ends cannot be approximated, or if there is a large defect between the freshened nerve ends, then nerve grafting is called for. All experimenters are agreed that the autograft is the best in animals, but that homagrafts and even heterografts may succeed in animal operations. The value of nerve grafting in man has yet to be determined by the results that will be obtained. Fresh grafts are preferable, a cutaneous nerve such as the cutaneous branches of the musculo-cutaneous or the internal cutaneous in the upper extremity and the long saphenous in the lower limb should be used. Whether especially good results in the human being would follow the use of grafts preserved in cold storage or in alcohol, has still to be finally determined. The results of the animal experiments justify a trial of these methods.[14]

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14. Huber, Nageotte: Compt. rend. Soc. de biol., 1918; Duroux et Couvreur: Lyon chir., p. 516, 1917. Dujarier et Francois: Bull. et mém de la Soc. de chir. de Par. **44**:43, 1918.

Huber's work, not yet published, is very complete and convincing, and I have been guided in my operations by the results of his experimental work. Lateral implantations and nerve plastics are not only useless, but directly harmful in the large majority of instances. The only proper nerve anastomosis is an end to end approximation, and with our present knowledge this is the only method of nerve suture that will give satisfactory results. Through the large number of peripheral nerve operations that have been and are being done, the finer technical details—into which we shall not go—have been thoroughly worked out, and there are today definite technical principles which must guide the surgeon in his operative work. The final results will not be known until several years have passed.

# Abstracts from Current Literature

THE MORPHOLOGY AND EVOLUTIONAL SIGNIFICANCE OF THE PINEAL BODY. Part I. Frederick Tilney, M.D., Ph.D., and Luther F. Warren, A.B., M.D., The American Anatomical Memoirs, No. 9, February, 1919. Published by the Wistar Institute of Anatomy and Biology, Philadelphia.

"Son siége au milieu de parties très-importantes de l'encéphale, sa constance chez l'homme et le vertèbrés, font pourtant présumer que ses usages, s'ils ne sont pas d'un ordre aussi important qu'on le supposait á l'époque des Esprits Vitaux, n'en sont pas moins réels et très-intéressant à connaitre." This introduction, taken from Legros, to a work of so complete, so critical and so finished a character as this first volume on the pineal body, suggests a suspicion of anti-climax which is, to say the least, not applicable to the volume which follows. The marshalling of the facts that are presented to us by these investigators through the comparative morphology, embryology and anatomy and histology of the pineal body in the various vertebrate types leaves for us no choice but to espouse their conclusion, which is practically forced on us. The morphologic problem has been formulated as follows:

1. What is the significance of the pineal region in its relation to the epiphysis?

2. Is the pineal body a vestige or is it an organ in some way necessary to metabolism?

3. Does its structure furnish evidence of its function?

4. What relation does it bear to the third or parietal eye?

5. What is the phylogenetic significance of the parietal eye with reference to the vertebrates and invertebrates?

Rather more than usual interest attaches to their explosion of the theory that the pineal body is the vestigial remains of the parietal eye. "The theory that the pineal body is the vestige of the parietal eye is accepted by many. According to this view, the third eye of vertebrates should be regarded as primordial and the pineal body an arrested development in the attempt to reach such differentiation. The evidence, however, is by no means conclusive, for, as has previously been shown, the entire epiphyseal complex springs from a region which is fundamentally glandiferous, while only in a very few instances is a tendency toward sensory differentiation recognizable in it. By far the great majority of vertebrates manifest in the epiphyseal complex no tendency whatsoever toward the development of any neural mechanism. This would seem to indicate that the tendency for the epiphyseal complex to develop visual structures is a secondary and not a primordial character. Furthermore, if the pineal body was in any true sense the vestige of the parietal eye, it would seem almost inevitable that the organ should contain remnants indicative of visual specialization. The absence of such evidence at least raises a reasonable doubt that the pineal body had at any time possessed visual function. The almost universal absence of true ganglionic cells as well as the lack of nerve fibers, which may be regarded as belonging to some category other than those of the sympathetic system, would seem to call into question

the possibility of the pineal body ever having participated in the formation of a neural mechanism. This may be considered negative evidence. There remains to be mentioned, however, the significant fact that the pineal body in all of the higher vertebrates manifests a tendency to differentiate along lines which cannot be interpreted as in the interests of visual function. As has been previously shown, the differentiation which does occur in the higher reptiles, birds, and mammals gives rise to glandular tissue. From these facts it seems possible to conclude that the pineal body is not a vestige of the parietal eye."

The answers to their questions are furnished as a conclusion and summary of the exhaustive analysis made by the authors, of the work of other investigators, as well as by their individual research; especially in the fields of comparative anatomy and histology and encephalic architectonic. These conclusions, respectively, to the questions propounded, are:

"I. The pineal region is preponderatingly glandiferous in its derivatives. The morphogenetic impulse imparted by such a gland-forming area could not fail to have a profound influence on one of its constituents, the epiphysis.

II. (*a*) The pineal body cannot be a vestige from the evidence based on its gross morphology, for the following reasons:

1. The phyletic constancy of the epiphysis in the vertebrate phylum.

2. Its variations and morphologic specializations.

3. Its relatively greater phyletic constancy with reference to other structures in the pineal region.

4. The gross evidence of its progressive specializations in ophidians, birds, and mammals.

5. The increase in the epiphyso-cerebral index, from the earliest stages to the latest periods of life in man.

6. (*a*) The resistance to the encroachment of a prominent neomorph in the mammalian brain, that is, the corpus callosum, which has produced such marked alterations in the other constituents of the diencephalic roof-plate.

(*b*) The pineal gland cannot be considered a vestige in the light of the histologic evidence, since the tendency toward specialization is definitely in the interest of glandular formation in ophidians, chelonians, birds, and mammals. The pineal body is, therefore, a glandular structure and as such, is necessary in some way to metabolism.

III. The histology of the organ gives clear evidence that the epiphyseal complex of vertebrates possesses a pluripotentiality whose fundamental inherent tendency is in the interest of glandular differentiation, but in a few instances, as in cyclostomes, amphibia, and in primitive reptiles, the pineal organ may become further differentiated in the interest of a highly specialized sensory mechanism which has, or has had, visual function. As a gland, it may in some cases, contribute its secretion to the cerebrospinal fluid, but in the higher vertebrates, as in ophidians, chelonians, birds, and mammals, it is an endocrinic organ, contributing the products of its secretion to the blood stream.

IV. (*a*) There is no direct relation between the parietal eye and the pineal body, but each is of itself an adaptive modification answering the demands for, or representing, an inherent impulse toward the development of a parietal eye, on the one hand, or a glandular organ, on the other.

(*b*) The pineal body as it appears in mammals cannot be regarded as the vestigial or metamorphosed degenerated or atrophic residuum of the parietal eye in vertebrates.

V. The phylogenetic significance of the parietal eye in vertebrates as the homologue of the median eye in invertebrates should be accepted with much reservation. Until such time as the homology between the vertebrate pineal region and some corresponding area of the invertebrate brain is much more firmly established than at present, the parietal eye as an index in the evolution of the vertebrates from the invertebrates has but little value."

It may be an interesting commentary in connection with the above conclusions that one of the authors, at least, approached the problem with a rather marked predilection against the view of the glandiferous character of the pineal body, and was converted to the endocrinous character of the conarium by the investigations herewith published. We look with anticipatory pleasure to the early appearance of the Second and Third Parts, embracing the physiology and pathology, and the clinical aspects of the pineal body. The reception of the work by comparative neurologists and by the endocrinologists should be of a most congratulatory nature to the authors, for it is an added foundation to the theory of the importance of the endocrine structures.

TIMME, New York.

CERTAIN OF THE CLINICAL ASPECTS OF "LATE CATATONIA" WITH A REPORT OF CASES. EDWARD A. STRECKER, M.D., Am. J. Insan. **74**:184, (Oct.) 1917.

In a study of 117 cases (two years' admissions) of individuals between 40 and 60, Strecker finds twenty-five cases in which catatonic-like symptoms existed. He concludes, however, that there is nothing to justify the assumption of late catatonic "disease process." The essential part of the paper is a study of the catatonic-like symptoms which he differentiates from true catatonic ones. He takes up various factors which may bring these about. He states that stereotypy may be simulated in some depressions in which the depressive affect is at a relatively low plane for a long time. In such cases the association between affect and affect-expression may become more and more indefinite, and when the affect disappears the expression may apparently be perpetuated in a purely automatic manner. A dementing process, "presumably arteriosclerotic," may then produce other catatonic-like symptoms such as spells of scratching, biting, kicking, etc., which are regarded as possibly irritative phenomena. Some refusal of food, resistiveness, etc., Strecker is inclined to attribute to an exaggeration of the "physiological" irritability of the climacterism. These symptoms, according to Strecker, often make the impression of outbursts of temper; they are therefore a reaction to the environment and associated with marked affects, and totally different from the more automatic catatonic manifestations. Confusion may give rise to resistance, struggling, etc., as a result of dangers confusedly seen in acts and occurrences in the environment. Such confused states, Strecker suggests, might be due to metabolism disorders. Somatic and nihilistic delusions are also a source of opposition, refusal of food, etc. As already stated, Strecker is inclined to attribute some influence to arteriosclerotic brain changes. It seems to the reviewer that Strecker is inclined to generalize too much about arteriosclerotic brain disease. He is, for example, inclined to attribute the bad prognosis of involution melancholias to this factor. There is no question

that there are cases with the symptomatology of involution melancholias in which a cerebral arteriosclerosis can be definitely demonstrated either by the characteristic deterioration (mental tension defect) or by focal symptoms. To go beyond this would seem unsafe and, in view of the distracting, absorbing affects, various apparent difficulties of thinking or orientation can often not even be used for this purpose. In general, while the reviewer agrees that there is no such "disease process" as late catatonia and that Strickler's interpretations are in the main acceptable, he feels that twenty-five cases are hardly enough to assume that no true catatonic symptoms occur, for there is after all probably no hard and fast line between, for example, the more automatic catatonic negativism and the opposition more clearly backed up by affects or ideas.

HOCH, Montecito, Calif.

ZUR DIAGNOSTISCHEN BEWERTUNG DER VARIETATEN DES BABINSKI'SCHEN REFLEXES. ROBERT BING, Basel, Schweiz. Arch. f. Neurol. u. Psychiat. **3**:89, 1918.

In a previous article Bing called attention to the fact that very often there occur certain associated movements of special muscle groups when the Babinski phenomenon is elicited. He classified them as follows: (1) Plain Babinski; (2) Babinski plus tonic hyperextension of the other toes or slow spreading (*fanning—signe de l'eventail*); (3) Babinski with simultaneous contraction of the tensor fascie latae, quadriceps or adductors of the thigh, and (4) Babinski with synchronous "flight or shortening reflex," that is, contraction of the tibialis anticus and flexion of the leg and thigh, lasting several seconds. It is Bing's opinion that significance can be attached to those variations of the Babinski and he takes exception to the view of Sussmann Galant that they are merely expressions of the various responses normally given by different individuals to a similar stimulus. The present article is based on a study of 246 cases showing undoubted spastic symptom complex of pyramidal tract involvement.

Bing noticed a striking difference of reaction in cases with pure cerebral involvement from those of purely spinal origin. In 101 cerebral cases he obtained the Babinski 79 times. Of these only 10 gave simultaneous contraction of the tensor fasciae, adductors or quadriceps, and not once did there occur the shortening reflex. On the other hand, in 46 spinal cases (out of a group of 56) which gave the Babinski he found 6 which showed simultaneous contraction of the thigh muscles and 24 the typical flight reflex, in all 30 cases, or 66 per cent. In cases with both spinal and cerebral involvement (M. S., C-S syphilis, etc.) he found 29.6 per cent. which gave the Babinski with associated contractions. In Bing's opinion the shortening reflex may be said to accompany the Babinski almost in every case in which there is compression of the cord of any kind. Of 28 proved cases only 7 showed exception.

As to the Gordon reflex Bing, in agreement with Auerbach, confirms its author's view that it is elicited more readily in early cases, while the Babinski in late cases, and that in compression of the calf it is a question of deep sensibility. He also noticed that the Gordon was obtained in nine out of ten cases of compression of the cord and that in the tenth case it was a question of syphilitic combined system disease. Knowing as we do that, *cessante causà*, recovery does occur in compression Bing agrees with Gordon and Auerbach that the reflex is elicited in cases with light pyramidal tract lesions.

The contralateral Babinski (or Oppenheim) the author found in six cases, all of which had unilateral cerebral lesions. The crossed reactions in these cases were of two types: (1) Dorsal extension of the big toe of the paretic side on attempt to elicit the phenomenon on the healthy side, and (2) double Babinski on stimulating the paralyzed side. Bing further remarks on the rarity of the Babinski phenomenon in amyotrophic lateral sclerosis. In none of the six cases which he studied did he find it. In six cases of "etat lacunaire" of the brain the Babinski was obtained five times, but always only on stimulation of the sole of the foot.

Bing does not draw any conclusions, but merely offers his observations for confirmation by other neurologists.

WECHSLER, New York.

SUR LE ROLE DE LA DYSTONIE DANS LA DESORGANISATION DES MOUVEMENTS VOLONTAIRES. (LOSS OF TONICITY AND INCOORDINATION OF VOLUNTARY MOVEMENTS.) W. VAN WOERKOM (Rotterdam), Nouv. Iconog. de la Salpêtrière **28**:37, 1916-1917.

According to the author's observations on a series of cases with anatomic lesions restricted to atrophy of the corpora striata and the region at the base of the optic layers, especially the Luys bodies, these parts may be regarded as exerting a considerable influence on muscular contraction, more particularly on the tonic element contained therein. Healthy individuals are enabled to utilize the regulatory function of the tonic factor in the performance of separate movements, other muscles of the body remaining in a state of tonic equilibrium. In these patients, however, the nervous impulse gives rise to a disturbance of the tonic element which sometimes involves nearly all the muscles of the body. This disturbance of the tonic factor of muscular innervation imparts itself in a great variety of forms. In spite of a very variable external appearance, characterized sometimes by choreiform seizures, in other cases by athetotic movements, or again by spasms and muscular rigidity, the similarity of all these manifestations is indicated especially by the part played by the factor of tonicity in the incoordination of voluntary acts, an entirely different character from that seen in cases with lesions of the posterior roots or the pyramidal tracts.

These patients are sometimes unable to initiate a simple voluntary movement. The concentration of the attention on the muscular movement often acts as an unfavorable element, preventing its accomplishment. Any muscular movement incomplete in itself can be much more readily carried out when it is a part of an habitual innervation complex, or when the attention is diverted. The disturbance exhibited by these patients at the beginning of a simple voluntary act is a very polymorphous and inconstant symptom, manifesting itself especially in the most elementary acts. The affected muscles are not paralyzed, the condition being rather a paresis of some of their functions, that is, a functional paresis instead of a true muscular paralysis. In the ultimate discharge of active movements, the disturbance of the regulatory functions imparts to the voluntary movements either an exaggerated, lively character, or a slow, irregular motion.

Dystonia manifesting itself in the incoordination of active movements often gives rise to innervation complexes approaching in part the picture seen in the classic contractures or in decerebrate rigidity, indicating the significance of the damaged parts for these functions. Lesions of the infracortical

centers, sometimes of the corpora striata alone, may accordingly cause a very peculiar interference with muscular function. The resulting disturbances of active movements present a certain resemblance to the cerebellar syndrome, but the necropsies showed no lesions of the cerebellum, the cerebellar peduncles, the red nucleus, the pyramidal bundles or the bands of Reil. The changes may be restricted to the corpora striata in the subthalamic region, but without involving the red nucleus. The cortex of the frontal lobes is occasionally the seat of evident changes, but in other cases is found to be intact.

GOODHART, New York.

DISTURBANCES OF VISION BY CEREBRAL LESIONS. GORDON HOLMES, C.M.G., M.D., Lieutenant-Colonel, R. A. M. C., Brit. J. Ophthal. **2**:353 (July) 1918.

Holmes first calls attention to certain conclusions regarding the cortical representation of the retina previously reached by himself and Lister. These are:

1. The upper half of each retina is represented in the dorsal, and the lower, in the ventral part of each visual area.

2. The center for macular or central vision lies in the posterior extremities of the visual areas, probably in the margins and the lateral surfaces of the occipital poles. The macular region has not a bilateral representation.

3. The center for vision subserved by the periphery of the retina is probably situated in the anterior ends of the visual areas, and the serial concentric zones of the retina from the macula to the periphery are probably represented in this order from behind forward in the visual cortex.

These conclusions are in conformity with those previously arrived at by Marie and Chatelin.

The author, after the examination of a large number of cases occurring in soldiers, in which there had been defects or disturbances of vision, produced by local cerebral injuries, has made certain observations which are of value not only in confirmation of the contentions already made but also in the elucidation of the probable representation of the different portions of the visual fields in the calcarine cortex.

First, there is taken up the question of local defects in the field of vision. Of these, central and paracentral scotomas are extremely common. Paracentral scotomas, especially in the lower quadrants, due to unilateral occipital injuries are relatively much more common than are central scotomas. In all the cases of unilateral inferior paracentral scotomas examined, the injury involved the tip or posterior portion of one occipital lobe, at or immediately above the level of the calcarine fissure, the size of the scotomas generally standing in close relation to the depth of the wound. Holmes presents three cases of the much less common superior paracentral scotomas. In these the lesion, as far as could be determined from the position of the wound and from roentgenographic examination, involved the lower part of the area striata.

In answer to the question as to why the area of central vision is so commonly affected in traumatic injuries, two explanations may be invoked. One is that the macula is projected on to a part of the visual cortex which is likely to be damaged by all injuries to the occipital cortex. The other is that macular vision, being very highly specialized, has a much more extensive cortical representation than has the peripheral. Both explanations are quite likely correct.

Four cases are presented showing sector scotomas. These cases tend to show, though in the absence of anatomic examination they cannot be regarded as conclusive evidence, that those portions of the retina which lie along the vertical radii are represented on the exposed visual surfaces of the occipital lobes, and by exclusion one might assume that the retina on either side of the horizontal radii is projected onto the visual cortex which occupies the walls of the calcarine fissures. A fact lending additional weight to this hypothesis is the great rarity of cases of sector scotomas for, if the hypothesis is correct it is what might be expected as it would be almost impossible to produce an injury to but a small area on the floor of the calcarine fissure.

Bearing on the cortical representation of peripheral vision and in accordance with a case recently described by Riddoch, is the case of a soldier, the roentgenographic examination of whom showed a piece of shell-casing just to the right of the midline in the position corresponding, in the average normal head, to the calcarine fissure immediately in front of the parieto-occipital fissure. Examination of this patient, on three separate occasions showed a contraction of the temporal periphery of the left visual field down to between 60 and 70 degrees and a slight contraction of the nasal half of the field of the right eye. A considerable number of cases in which quadrantic hemianopias or other regular defects in the fields resulted from lesions of the optic radiations were seen by the author.

In two cases reported, in which direct injury of the calcarine cortex can be excluded, lesions of the lower parts of the optic radiations of one side produced a superior quadrantic hemianopia. These findings together with those of other cases cause the author to admit that it is tempting to assume that in the main mass of the optic radiations the fibers connected indirectly with the upper and lower halves of the retina are contained in distinct bundles separated from each other by an anatomic interval. This hypothesis, however, cannot be accepted without further evidence, it is felt. No case has been observed in which a paracentral scotoma, or an isolated affection of central or pericentral vision, could be attributed to a lesion of the radiations.

Regarding the portion of the field in which recovery first occurs, Holmes takes exception to the statement of Riddoch that "the recovery of the appreciation of movement begins in the periphery of the field and extends inward toward central vision." Both his own cases and those of Hine tend to disprove this.

Many of the transient and recoverable symptoms are attributed to degeneration of the myelinated fibers and especially to edematous swelling. Holmes is also of the opinion, as far as his observations go, that the so-called dissociation of visual perception, due to occipital injuries, recently described by Riddoch, should not be spoken of as a dissociation of the elements of visual sensation, since it is only a condition of visual hypo-esthesia in which only the stronger and more adequate stimuli excite sensations. It is admitted, however, that dissociation of function, with intact retinal sensibility, may be caused by injuries of other parts of the brain. Observations extending over a large number of cases have also tended to show that an isolated loss or dissociation of color vision is not produced by cerebral lesions.

Two unusual affections of vision, occurring in lesions of portions of the brain anterior to the occipital lobes, have been noted. The most frequent of these is an affection of visual attention. In this condition, though there is no diminution of visual sensibility or restriction of the fields, the patient frequently fails to recognize objects brought into the affected homonymous

halves of the fields, though they are perceived at once when introduced into the opposite halves. This condition is usually associated with lesions of the parietal lobe involving the supramarginal gyrus or its neighborhood. In some of the cases with this condition there was noted a similar local tactile attention loss, of the type previously described by Head and the author, on the same side of the body.

The author has seen six additional cases of a form of visual disturbance of the type previously reported by him with Smith, in which there is a loss of visual orientation and appreciation of depth, in which the condition is seemingly dependent on injury of the brain in the neighborhood of the angular and supramarginal gyri.

This valuable and interesting article is summarized in the following conclusions:

1. Those portions of the retinae adjoining their vertical axes are probably represented in the dorsal and ventral margins of the visual areas, while the retina in the neighborhood of its horizontal axis is projected onto the walls and floor of the calcarine fissures.

2. Severe lesions of the visual cortex produce complete blindness in the corresponding portions of the visual fields, or if incomplete an amblyopia, color vision being generally lost and white objects appearing indistinct, or only more potent stimuli, as abruptly moving objects, may excite sensations.

3. The defects of vision in the fields of the two eyes are always congruous and superimposable, providing that no abnormality of the peripheral visual apparatus exists.

4. Lesions of the lateral surfaces of the hemispheres, particularly of the posterior parietal regions, may cause certain disturbances of the higher visual perceptual functions with intact visual sensibility, as loss of visual orientation and localization in space, disturbance of the perception of depth and distance, visual attention loss, and visual agnosia.

STANTON, Ann Arbor, Mich.

THE THEORY OF SYMBOLISM. ERNEST JONES, M.D., Brit. J. Psychol. **9**:181, (Oct.) 1918.

In this considerable discussion of symbolism Jones has both extended and further specified those notions which emerge from the more strict freudian school as contrasted with the ideas of Jung, Silberer and others whom, with, perhaps, not a well timed animus, he has termed the "postpsycho-analytic school." His own argument runs so well on all fours that there is even less need for this feeling. There can be no doubt that the stimulus afforded by the Zurich school has had an effect within the more strict freudian circle. Yet the extensions in the later departures of Jung contain much sufficiently mystical, religiostic and ethical to give point to the clear and psychologic criticism effected in this article by Jones. It is this aspect which here will be briefly presented.

His isolation of symbolism, as a fundamental type of indirect representation, from others more or less allied, as metaphor, etc., is preceded by a consideration of symbolism in its broader sense as indicating "a relative incapacity for either apprehension or presentation," affective or intellectual in origin, the former being the more important. This incapacity is not a cause, as Silberer makes it, but an indispensable condition. Because of this inca-

pacity the mind reverts to the simpler type of mental process, with degrees in this regression, relating the more highly developed forms of symbolism to those points where the inhibition of feeling is greatest.

Through ths mode of approach by way of the more general symbolism Jones raises the question as to how in relation to the inhibited feeling there may be seen the function of the metaphor in which an adjective or connotation is replaced by a concrete likeness. This method of approach to the subject appears useful in view of our well known resistances. He defines identification as the basic feature of symbolism and related both to the pleasure and reality principle. To the first in the fact that it is more pleasant to read the new by way of the old, for the interest resides here; that is, it is not a question of immature development and failure to comprehend on this basis which drives to symbolism, but a dynamic interest in pleasure which guides to this end. He rightly apprehends the effect of this kind of development along affective and intellectual lines.

Where the affective inhibition is highest, symbolism in its typical form appears, and for this Jones proposes to reserve the term of symbolism, although he recognizes the continuum between this and other forms. He specifies this position, then, as making symbolism that in which the process is unconscious, with the affect investing the symbolized idea and further as being "not capable of that modification in quality termed sublimation," to which latter term he has given also a better meaning by seeing in it an "ontogenetic repetition of the deeper tendency of phylogeny of draining off sexual energy into non-sexual channels." It is hence more biologic and as a concept has less of the conscious moral or ethical which is improperly imputed to it.

The typical attributes of true symbolism are (1) representation of unconscious material; (2) constant meaning; (3) nondependence on individual factors only; (4) an evolutionary basis; (5) linguistic connections between the symbol and the idea symbolized, and (6) phylogenetic parallels. The ideas symbolized "arise as the result of a regression from a higher level of meaning to a more primitive one; the actual and real meaning of an idea is temporarily lost and it is used to represent and carry the meaning of a more primitive idea, with which it was once symbolically equivalent." The notions involved here are important, for the difference of most pragmatic value established by Jones, in opposition to Jung and Silberer, is defined in the notions of progression beyond this early stage of symbolic equivalency. A different attitude taken here yields a totally different orientation as to individual progression, a matter of large moment in the consideration of adaptations and maladaptations. Progress ensues beyond the early stage of "symbolic equivalency" where the symbolic meaning has been transferred from the primary, or unconscious, idea to a secondary idea going along with the modification of the affects belonging to the primary idea, to the end that these affects may become attached to more useful conscious and noninhibited ideas.

There is here a partial renunciation as regards the original complex, with, however, a compensation of it by other ideas. But if the affect of this complex, or primary idea, be not considerably modified, the complex pushes forward, not in its original form, but by means of its early symbolic equivalents, like a secondary idea. This latter, then, carries a significance of the complex and becomes its symbol. Beyond this there can be no progression until there be a further loosening of the bonds between the complex and its substitutive secondary idea, or symbolic equivalent, with the more definite renouncing of the complex as a source of direct gratification. Jones emphasizes in this fact

that by way of the symbolic equivalents progress is made. It is the symbolic equivalent that is the basis of symbolism. "Progress does not take place via the symbolism, but via the symbolic equivalents; the symbolism itself in fact constitutes a barrier to progress. This is best seen in the blind alley of neurotic symptomatology."

Jones sees Silberer's failure as the "confounding of the process of symbolic equivalency with that of symbolism itself." Jung and Silberer have described progress by way of the symbols. The notion of the "future idea," "the higher form of truth," as residing in the symbol is thus denied by Jones, who sees in the continued "symbolic use of the symbol" that function which prevents truly progressive ideas from occurring. It is only by a surrender of the subjective that the higher objective meanings may come about. He expands this by considering progression as seen in other conscious tendencies, as science, there differing from the symbol use "in that with the latter the original unconscious complex is retained unaltered and merely transferred on to a secondary idea, that of the symbol, while with the former the psychical energy alone, not the significance, is derived from the unconscious complexes and is transferred on to another set of ideas that have their own significance."

Silberer's "functional interpretation" of the symbol Jones considers as "the regression proceeding only a certain distance and as mainly concerned with the more conscious reactions to the sublimation of the unconscious complex." Silberer, then, confounds really that which is more like a metaphor in structure with that of true symbolism which represents regression to a deeper level. The difference is one between "over-sublimation in the metaphor and under-sublimation in the symbolism." That is, Silberer, as Jones sees him, locates the meaning of a symbol less in the unconscious complexes and more in the ethical repressions effecting this unconscious and in the sublimated tendencies issuing out of the unconscious dynamics. While the material of the symbol, Jones holds, is derived from these sublimations, the meaning of the symbol is drawn totally from the unconscious; nor can it be located in the repressing factors which in their effect on the unconscious have brought about the sublimations. That the sublimations and repressive factors may be symbolized, Jones grants, but it is here a metaphor; there is used a borrowed symbol for another end. That is, the unconscious is the original giver of meaning to the symbol; if another meaning be given to it it is metaphorical or functional.

The entire essay is worth study for a consideration of the psychologic mechanisms so acutely defined in symbols, in their isolated setting affording a somewhat sharper outline than when presenting in the psychoneurotic and psychotic. It affords also an orientation as to the better reconstructive lines to be pursued and those most aptly coinciding with the evolving modes of the individual. The sharp counterpoising of two apparently opposing points of view enhances the value for the reader.

PARKER, New York.

PARALYSIE GENERALE CHEZ UNE OXYCEPHALIQUE. (GENERAL PARALYSIS IN A PATIENT WITH AN OXYCEPHALIC SKULL.) H. FLOURNOY, Nouv. Iconog. de la Salpêtrière, **28**:15, 1916-1917.

The author's patient was a woman, aged 34, with a skull marked by oxycephalus—having a sharp peaked crown and a high vertical index. The patient entered the hospital suffering from general paralysis and died about six weeks later as the result of pulmonary embolism.

Aside from all etiologic considerations, oxycephalus is attended by certain sudden manifestations which the author is inclined to attribute to the abnormal curvature of the aqueduct of Sylvius, causing sudden ventricular stasis comparable to the symptoms of some brain tumors. It is possible that oxycephalus may be related to an irregular periodical increase in size of the brain, not involving all parts of the organ at the same time. The optic nerve atrophy seen in these cases is not the result of a narrowness of the bony canal, but is due to an exaggerated intracranial and intraventricular pressure, together with a damming-back of the cerebrospinal fluid which results therefrom. Paralysis of the third cranial nerves is also dependent on the intraventricular pressure; it is a nuclear paralysis, the nucleus being situated under the wall of the third ventricle and the floor of the aqueduct.

One of the most distinct postmortem signs of pressure is the existence of digitated impressions on the internal surface of the skull. The erosion of the bone may reach such a degree as to terminate in spontaneous perforation. The maximum of these digitated impressions corresponds to the slightest thickness of the cerebral cortex and the cranial vault.

GOODHART, New York.

THE TREATMENT OF SECONDARY AND LATE COMPLICATIONS OF BRAIN WOUNDS. Comptes-Rendus de la Conférence chirurgicale interalliée pour l'étude des plaies de guerre, 3e session, 5-8, Novembre, 1917. GORDON HOLMES, Editorial Comment, Ann. Surg. **69**:184 (Feb.) 1919.

Holmes reviews the records of 2,357 patients admitted to the hospitals of the United Kingdom with gunshot wounds of the cranium. Of these, 1,567 dated back far enough to study the secondary complications—from three months to two or three years.

*Brain Abscesses.*—These developed in thirty-seven cases. The great majority occurred between the third and sixth month. From then on the number of cases rapidly diminished, and only one case occurred after the tenth month. Most frequently they developed around bony sequestra or under necrosed fragments of the cranium. In a small percentage of cases they developed around retained foreign bodies. Abscess is relatively more frequent in wounds involving the orbit or ethmoid region. Of the thirty-seven cases, most of them were operated on, but the operative results were not satisfactory. Twenty-eight cases terminated fatally. The best method of treatment is puncture or evacuation of the abscess by small incision followed by drainage.

*Meningitis.*—Primary meningitis is the most serious complication and most frequent cause of death in the first period of brain wounds. It is relatively rare in the later course of such wounds, occurring chiefly secondary to abscess.

*Encephalitis.*—The clinical differentiation between encephalitis and abscess is very difficult. Cases sometimes clear up after the extracton of superficially placed foreign bodies, sometimes after a simple exploratory operation, and sometimes after rest and an ice cap.

*Infection of Ventricles.*—This occurs most frequently in cases of hernia cerebri with much loss of substance. The ventricle forces its way into the protruding brain tissue and becomes directly infected. Rupture of an abscess into the ventricles leads to the same result. In the former case recovery may occur. The latter is always fatal.

*Hernia Cerebri.*—This is generally an early complication. But it may persist long, or it may develop late. Of ninety severe cases, seventy recovered,

so that the outlook is not as bad as it at first seemed. The best treatment is the application of aseptic or antiseptic dressings. Surgical treatment properly so-called, is almost uniformly unsuccessful. However, bone splints and fragments of foreign bodies should be removed, and pus collections drained. Operation should be strictly limited to the inflamed parts, and the rest of the brain carefully protected. Contra-lateral decompression sometimes gives good results.

*Epilepsy.*—Attacks of general or jacksonian epilepsy are very frequent in the earlier period of wounds, but persist in only a small percentage of the cases. Frequently they disappear after the escape of pus or the subsidence of inflammatory foci. It is impossible as yet to estimate the percentage of epilepsy as a later complication of head wounds; but it is less frequent than was formerly believed. Epilepsy occurred in some cases of simple concussion, namely, in the absence of fracture or depression. As to treatment, aside from the removal of a foreign body or the clearing of an abscess, surgical intervention is practically useless. The disease is probably most often due to the presence of cicatricial tissue. In some hospitals it has been the routine to administer bromids to all cases of head wounds, and it would seem that this had lessened the frequency of the complication.

*Mental Troubles.*—Slight disturbances, such as depression, irritation and forgetfulness are common. Severe mental troubles are rare, and are most frequent following extensive frontal wounds.

*Extensive Losses of Cranial Wall.*—Aside from the symptoms common to all cranial wounds—headache, vertigo, amnesia, inability to concentrate and insomnia—no special symptoms arise from cranial defects. The principal inconvenience is the danger of injury to the unprotected brain. This can be remedied by protective plate or graft.

*Secondary Effects of Retained Splinters.*—One hundred and sixty-four cases with retained fragments were examined after from three months up to two or three years. Twenty-three had returned to active service; 129 had been invalided, but of these, thirty-six had taken up some civil occupation; twelve patients died—two following the shock of operation for the removal of the foreign body. Abscess was the most common secondary complication. The average mortality of these cases was 7.3 per cent. as compared with 4.7 per cent. for all cases followed after three months. There is no doubt that in the majority of cases, balls or fragments of shrapnel may remain in the brain without producing accident or symptoms if they are not septic. Considering the danger of attempted removal where the fragments are deeply placed they should be left alone unless showing symptoms of irritation or progressive cranial disease.

SELLING, Portland, Ore.

COMPARATIVE STUDIES ON THE GROWTH OF THE CEREBRAL CORTEX. NAOKI SUGITA, M.D., J. Comp. Neurol. **28**:495 (Dec.) 1917; **29**:1, 11 (Feb.) 1918; ibid. **29**:61 (April) 1918; ibid. **29**:177, 241 (June) 1918.

In the program of the neurological laboratory of the Wistar Institute the problem of the growth of the mammalian nervous system, between birth and maturity, is given the first place.

Some years ago this work was begun by the study of the growth changes in the larger divisions of the nervous system—the brain and spinal cord—and since then this study has been extended to the subdivisions as occasion offered.

Studies of this sort, which deal with size and number, meet at once the technical difficulty arising from the changes in size produced by the reagents employed for the preparation of the material.

Dr. Sugita has overcome this difficulty by determining in every case the amount of change caused by the reagents employed, and by this means has computed correction factors by the aid of which all the measurements can be referred to the fresh specimen. This procedure has not been used before and it therefore makes these studies in a certain sense unique, while at the same time it serves to show the precautions necessary in such determinations —for without these precautions the results would be valueless.

It was Dr. Sugita's aim to determine by measurements how the cerebral cortex grew in the domesticated albino rat; whether its growth in the wild Norway was similar to that in the Albino, and also how far the results for the rat agreed with those for man.

To obtain these facts he has followed, in both the Albino and the gray rat, the changes in the shape of the cerebrum with age (I and III); the increase in the thickness of the cortex (II and IV); the increase in the area and volume of the cortex and in the computed number of nerve cells in it (V, Parts 1 and 2); the growth of the nerve cell bodies (two groups) in the cortex (VI, Parts 1 and 2); the influence of early starvation on cortical growth (VII) and finally, he has made a comparison of his results on the rat with those reported for other mammals (VIII).

From the series of observations on the two forms of the rat and the several comparisons which they permit, a number of general relations appear, as follows:

The longitudinal diameter of the cerebrum tends to become relatively longer during growth (I and III). The thickness of the cortex is greatest at the frontal pole and on the dorsal surface and diminishes gradually, caudad and ventrad. The cortex attains nearly its complete thickness (96 per cent.) at twenty days—the age at which the young rat is weaned. However, at twenty days the brain has only a little more than half its final weight, so that the extension of the cortex to cover the larger cerebrum of the mature brain is accomplished without any significant change in its thickness. At twenty days the computed number of cells in the cortex is complete, but this number is about twice that found at birth, showing that cell multiplication and the migration of cells into the cortex have been active, but have come to an end at the weaning time of the rat.

During the first twenty days of life the enlargement of the cell bodies runs parallel with that of the cortex as a whole. The cell bodies in the pyramidal layer and in the ganglion cell layer do not, however, mature (in size) at the same time, the latter being somewhat in advance of the former, and further, the pyramidal cells after reaching their maximum show clearly a tendency to diminish in size.

When the data for the wild Norway are compared with those for the domesticated Albino they are found to agree in the time relations. Quantitative differences, of course, appear, for we know that at a given age the brain of the wild Norway is both absolutely and relatively heavier than that of the Albino. In accordance with this relation the cortex in the Norway brain is thicker and has a greater volume, and the cells composing it are larger. On the other hand, it is shown that the computed number of the cells in the cortex is the same in both forms—an important result. The number of cells is therefore characteristic for the species.

Moreover, the Albino differs from the Norway by the fact that the eyes in the Albino are imperfect, and vision is poor.

As might be expected, therefore, when a comparison of the thickness of the occipital cortex (visual area) is made, it is found that the cortex in the Albino is especially thin in that region.

When young rats have been starved during the suckling period the effect is to reduce the growth of the cortex by reducing the size of both the cells and fibers, yet the number of the neurons remains unmodified (VII), thus adding to the evidence that each mammalian species probably has in its nervous system a characteristic number of neurons—a number which is constant within the limits of biological variation.

So far as comparisons could be made there appears to be a similarity in the growth of the cortex in the several mammals studied by other investigators, and the scanty data for man show that the phases of growth in the human cortex are similar to those in the rat at the equivalent ages (VIII). The cortex in man therefore has probably attained nearly its complete thickness at the age of 15 months.

DONALDSON, Philadelphia.

# Society Transactions

## PHILADELPHIA NEUROLOGICAL SOCIETY

*Regular Meeting, Feb. 28, 1919*

J. Hendrie Lloyd, M.D., *President*

TWO CASES WITH ATTACKS OF SOMNOLENCE, PROBABLY OF PITUITARY ORIGIN. Dr. B. P. Weiss presented these cases.

Case 1.—*History.*—The patient is a trainman, aged 41. His chief complaint is periodic somnolence which is uncontrollable. Family history is negative. He denies syphilitic infection. Uses tobacco moderately; no alcohol.

His present illness began about one year ago, when patient would unexpectedly fall asleep. This sleep was profound but would only last a few minutes. Says his head falling on his chest usually awakens him. These periods of somnolence occur only while he is on the train. He has gone from fourteen to sixteen hours without falling asleep once, while at other times he might have from three to twelve attacks during a "run" of fourteen hours.

*Physical Examination.*—Eyes: Pupils equal, regular, react to light and accommodation. Eye grounds normal. Subcutaneous tissue seems somewhat boggy. There is no cranial nerve involvement. No ataxia of the limbs. There are no tremors and the grip is good. The biceps and triceps reflexes are normal. Gait and station normal. The knee jerks and tendo-Achilles jerks are present and equal on both sides. Ankle-clonus and Babinski reflexes are absent. Sensation is unimpaired. The mental reactions are somewhat sluggish. He is rather slow in answering simple questions. The memory is slightly affected.

*Laboratory Reports.*—Spinal Fluid: Ten c.c. clear fluid was removed. Pressure not increased but ran freely. Globulin, plus 1; cells, 6; sugar, positive; Wassermann test, negative; blood Wassermann, negative. He was given 200 gm. glucose dissolved in tea. The entire urinary output for the next ten hours was saved; it did not reduce Fehling's nor Nylander's solutions. The urinalysis was negative.

*Roentgen-Ray Report.*—There is enlargement of the sella turcica with some change in the posterior clinoid processes, probably bony deposits. Plates of sinuses show perfect clearness.

Case 2.—*History.*—The second patient is a salesman, aged 27. His chief complaint is somnolence, snoring and nocturnal polyuria. His parents are first cousins. There was no nervous disease in family. He is married and has one healthy child. No miscarriages. Uses no alcohol. Had smoked twenty cigarets daily, but lately has been more moderate. Never had a chancre. Always been fat. Average weight 175 pounds. About a month ago he had the grippe and lost 30 pounds. During this infection, which lasted about two weeks, attacks of somnolence occurred only at very infrequent intervals and were of very short duration.

His present illness started with attacks of somnolence while at work. This has increased so that now he goes to sleep sitting and even while standing. Sleeps continuously from twenty-four to thirty-six hours if undisturbed. He cannot control the attacks, even if in the midst of noise, or while listening to a band the sleeping state will occur. At times while asleep respiration stops for a minute or two, then it becomes rapid and irregular, accompanied by considerable respiratory difficulty, and choking attacks. From the description given by his wife, respiration during sleep resembles the Cheyne-Stokes' type. Polyuria is nocturnal only. Sometimes he passes 3,000 c.c. during a night, even while on a diet with four glasses fluid per day.

*Physical Examination.*—An obese, short, young, adult male, rather sallow. Hands and feet small, fingers tapering, skin dry. Subcutaneous tissue appears to have a mucoid consistency. Eyes: Pupils react normally. No muscle imbalance. Eye-ground examination reveals nothing of moment. Thyroid palpable. Station and gait normal. No alteration in the knee or ankle jerks. Ankle clonus and Babinski sign are absent. Sensation is unimpaired.

*Laboratory Reports.*—Blood Wassermann: negative. Spinal fluid examination shows no pathologic change. Urinalysis was negative. Sugar tolerance: when given 200 grains of glucose, no sugar was eliminated in urine.

*Roentgen-Ray Report.*—Sella turcica is comparatively small. No other evidence of lesion.

DISCUSSION

Dr. Francis X. Dercum said that these cases were of some clinical importance, that they were met with every once in a while and frequently were not recognized. They represented internal secretion disease. In some of these cases Dr. Dercum has gotten good results from thyroid administration. His impression was that the smaller of the two men—the one with pointing of the fingers and the retardation of growth suggesting pituitary deficiency—also suffered from sexual weakness. This man improved for a time on a combined thyroid and pituitary treatment.

Dr. B. P. Weiss stated that there were a number of inquiries concerning the sexual apparatus. The second patient presented some deficiency in the sexual apparatus, but his function was unimpaired. He was married, his wife had had one child and no miscarriages. The taller man had no alteration in his sexual make-up and his function was unaltered.

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TRAUMATIC BRACHIAL PLEXUS PALSY, OPERATION—MARKED IMPROVEMENT. Second presentation of this case by Dr. F. X. Dercum.

*History.*—L. H., aged 24, had been shown at the October meeting of the Society. At that time he presented a severe palsy of the right upper extremity. He had the following history: On April 19, 1918, while standing at the Dupont Powder Works near a press loaded with 60 pounds of gun cotton, the latter suddenly exploded. He was thrown into the air and fell on his back on a table some 10 or 12 feet away. He was unconscious for a few minutes and, when he came to, found himself lying on his back with his right arm under him. He was able to raise himself and to walk a distance of about 150 feet and was then removed to the hospital of the Dupont Works in an automobile. At the hospital he was examined by Dr. Stocks. It was found that the right

arm was paralyzed from the shoulder down; there were also a fracture of the metacarpal bone of the right thumb and several burns, bruises and abrasions over the left arm and wrist. The patient states that the right arm was not only entirely helpless, but also without feeling; he had no pain in the right arm or shoulder, but there was considerable pain in the left arm.

*Examination.*—On May 4, 1918, when first examined by Dr. Dercum the right upper extremity presented a flaccid palsy, with loss of reflexes, involving the hand, the forearm, the upper arm and the deltoid. The patient could shrug the shoulder, but no movements whatever of the arm itself could be performed. No pain or tenderness could be elicited over the brachial plexus or over any of the nerve trunks. Sensation was entirely lost over the hand and forearm; on the upper arm it was slightly preserved to all forms on all aspects; on the inner aspect of the arm it was slightly better preserved than elsewhere and extended for a short distance below the elbow on the forearm. On the anterior, outer and posterior aspects, sensation though diminished was preserved as far as the junction of the upper and middle thirds of the arm.

On June 25, he was admitted to the Jefferson Hospital. Marked wasting of the muscles of the hand and forearm and to a slightly less pronounced extent in the muscles of the upper arm and shoulder had been established. An electrical examination now revealed faradic and galvanic extinction in the muscles of the hand, forearm and upper arm. The deltoid responded equally to KCl. and AnCl. There had been no changes in the areas of sensory loss. In the outer aspects of the upper arm it was somewhat fainter than at previous examinations. A roentgen-ray examination proved negative. A few days later (July 1) the brachial plexus was freely exposed by Dr. J. Chalmers Da Costa. It was found that the plexus was firmly embedded in scar tissue and its various cords were separated with considerable difficulty. No gross loss or break of continuity was observed. It seemed probable that the plexus had been the seat of a severe contusion with possibly marked extravasation of blood.

*Clinical Course.*—Little or no change was noted in the condition of the arm for a long time. It was, however, faithfully rubbed, well supported and not allowed to drag at the shoulder. At the first presentation of the case in October, little change was noted; save that Dr. Dercum thought there was a faint return of power in the deltoid and also that there had been some improvement in the sensation of the upper arm. It seemed to descend to a slightly lower level. Several members who were present were not inclined to concede either the action of the deltoid or the presence of sensation on the outer aspect of the arm. The presence of sensation on the inner aspect was clearly due to the intercosto-humeral nerve.

During the winter, the arm has been steadily treated by massage and electricity. At present (Feb. 28, 1919) a remarkable degree of improvement is evident. The arm can now be extended in a horizontal line at the shoulder. There is a feeble—a beginning—return of flexion and extension at the elbow. There is undoubted action of the biceps and triceps. There is a feeble movement of pronation due to the action of the biceps. There is no movement at the wrist or in the hand. Sensation is preserved for touch and pain throughout the upper arm except for a small area on the outer and lower surface of the arm. Sensation is lost below the elbow except for a small area posteriorly and internally just below the elbow.

The power to elevate the arm is no doubt reenforced by the synergic action of shoulder muscles other than the deltoid, but the return of power in the

deltoid though not great, Dr. Dercum thought, must be conceded. The prognosis was at first most discouraging as it seemed not improbable that the plexus had been most extensively injured, perhaps even torn from its roots. The very marked improvement, however, justifies the hope that it has not yet obtained its maximum but will continue.

DISCUSSION

Dr. T. Turner Thomas said that he had looked particularly for relaxation of the shoulder joint. He did not find it. He looked again particularly for ankylosis of the shoulder joint, or limitation of movement, and he found that the patient had power to raise his arm as far as the joint movement permitted it to go. Dr. Thomas had observed about ten years ago that some of these cases of what were thought to be traumatic brachial paralysis disappeared with the return of normal function of the shoulder joint, excepting when there was a relaxation of the shoulder joint and in every one of those instances in which he operated to restore the shoulder joint to its normal firmness and relations power returned. He came to the conclusion that in the cases that he saw there was not an actual rupture or injury of the brachial plexus, but that probably as the result of the injury to the shoulder joint in most cases there was an extravasation of blood, perhaps synovial fluid, around the nerves adjacent to the injured shoulder joint extending upward to the plexus. He had never been able to prove that by exposure of the plexus, because he believed such operations were not necessary, the extravasation disappearing by absorption. Dr. Thomas stated that Dr. Dercum's patient represented a very severe case. He could not recall one in his experience in which the hand had been so helpless. He had read Madame Klumpke's paper carefully and he had come to the conclusion that the adult cases which she reported were not of this type at all, that they were really brachial plexus ruptures. They were cases in which there was generally a complete loss of power and of sensation, usually below the elbow. In none of his cases was there such a complete loss of power and sensation. Rarely was there much loss of sensation. In some there was almost complete loss of power throughout the whole extremity. In most of them the power in the hand remained and was better than in any other part of the limb. Dr. Thomas' interest in this case was largely in the fact that operation failed to show actual rupture of the plexus. Operations have been done on obstetrical brachial palsies and reports have varied a great deal. He said he had not been willing to accept the findings of ruptured nerves because they had been findings in dense cicatricial tissue and he thought that dissecting nerves out of dense cicatricial tissue did not allow one to make very accurate observation, but in most of the cases no ruptures of the nerves were found, and he thought at the present time that operation on the plexus in obstetrical palsies had nearly gone out of vogue.

Dr. William G. Spiller said that the recovery of power in the brachial plexus when the loss had been so intense was interesting, and yet he felt that the man illustrated in addition to recovery of power what is sometimes seen in long-standing partial palsy, a compensatory action of other muscles replacing in some degree the imperfect function of the weak muscles. The man threw his shoulder backward before he attempted to raise the upper limb, and in this way secured a better position for the action of the supraspinatus and possibly of the trapezius. The case taught that the most desperate palsy may not be hopeless. He had seen this illustrated recently by an intense

ulnar nerve palsy. The paralysis had developed as a result of tying the upper limbs to the bed during a delirium. The palsy and atrophy could not have been more intense, and yet after a few months of electrical and other treatment the hand had almost entirely recovered.

Dr. Francis X. Dercum, in closing, said that when he first saw the case he thought of Dr. Thomas' operation. He did not find that the capsule was relaxed. The signs of an actual nerve palsy were pronounced. On exposure of the brachial plexus it was found matted together by dense cicatricial tissue. Dr. Dercum had not expected the improvement which had followed. He thought that the deltoid contraction could be felt under the finger.

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ENCEPHALITIS SIMULATING BRAIN TUMOR. Presented by Dr. D. E. Smith.

*History.*—E. S., aged 16 years, was admitted to the University Hospital, Jan. 15, 1919, to the service of Dr. Spiller. He was entirely well until Nov. 1, 1918. At this time he began to have a heavy, dull feeling located in the frontal region, followed by a rather severe headache. This condition lasted about thirty days and was always intensified by any work, and became gradually more intense until about November 20 and then subsided. During most of this time there was nausea, with frequent projectile vomiting. He vomited whether he took food or not. He was not unconscious and had no convulsions. A period of improvement followed from Dec. 1, 1918, to Jan. 8, 1919, there being an occasional attack of headache and vomiting, but of less severity. On Jan. 8, 1919, he was seized with intense headache, drowsiness, nausea and vomiting and was unable to obtain relief. Sleeplessness was caused by the pain. His eyes ached intensely. He occasionally saw objects double. He may have had influenza before the first attack, but this was uncertain. The laboratory findings for syphilis were negative.

*Physical Examination.*—This was entirely negative except the following: Jan. 17, 1919: vision, O. D., 6/7.5; O. S., 6/15. The pupils were equal and normal. The left superior oblique muscle showed paresis. The media were clear; the disks were very hyperemic, the margins obscured, and the disks were moderately swollen. The retinal veins were dark and tortuous. O. D. disk level was +3 D., fundus +1.5 D. O. S. level was +2 D., fundus +1 D. Diplopia was found over the entire field. Lower fields full; some contraction concentrically of the color fields (Dr. Langdon). Dr. Grayson found complete anesthesia of the soft palate, and the soft palate was also paretic. The anesthesia extended on the left side of the pharynx to about the median line. On February 27 the palatal muscles were still sluggish, but distinctly less paretic. The sensibility also was increased on the left side of the palate and pharynx, though much less so than on the right side. On February 28 no diplopia and no muscular paresis were reported. There had been no fever of any moment, the highest being 100.4 F. once; at times it was subnormal. The pulse was full and regular, no bradycardia.

The ocular findings were important and were obtained at short intervals. The ocular condition became gradually worse and on February 13 vision of the right eye was 6/9, of the left eye 6/15. The right disk was +5½ D., the left +5 D. Lumbar puncture reduced the swelling promptly about 1 diopter, and as frequent examinations showed the swelling of the disks was

subsiding decompression was not done. On February 28 vision of O. D. was 6/6, of O. S. 6/12. The right disk was + 3½ D., the left + 2½ D.*

DISCUSSION

Dr. William G. Spiller said that this boy when he first came under his care presented symptoms which were strongly suggestive of intracranial tumor. It was difficult to explain by a diagnosis of tumor the paralysis of the left superior oblique muscle and the paresis and loss of sensation of the soft palate and loss of sensation of the left side of the pharynx. While it is true that many axis cylinders pass through a glioma and that the symptoms of such a tumor are much less intense than the size of the tumor would indicate, it seemed improbable that a glioma could be extensive enough to cause the disturbances of function mentioned without other symptoms. A lumbar puncture done on January 19 had given 3 or 4 cells to the cm., and the Wassermann and globulin reactions were negative.

The diagnosis of a diffuse process was made, and encephalitis was considered. The boy had had a little fever. Serous meningitis was a possible diagnosis. Dr. Spiller had had lumbar puncture done to see the effect on the swelling of the optic disks, and the puncture had caused a rapid diminution in the height of the swelling. He had postponed a cerebral decompression, as repeated observations showed that the condition, including that of the eyes, was improving. The boy is now well.

Dr. Francis X. Dercum asked how long-standing were the symptoms before the boy came under observation.

Dr. Smith replied about two and a half months. Dr. Dercum thought that a possible explanation was offered by serous meningitis.

Dr. Alfred Gordon said the case was an exceedingly interesting one from the standpoint of diagnosis. He recalled another case in which the patient complained of severe headache and had projectile vomiting. The vomiting was so severe that she could not sleep and the patient had distinct asynergia; the knee-jerks were markedly diminished. Everything pointed to possible cerebellar involvement. The Wassermann examination was negative. After the removal of about 20 c.c. of the spinal fluid the patient began to improve and made a complete recovery.

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## BRAIN TUMOR WITH EXACT ROENTGEN RAY LOCALIZATION.

Presented by Dr. J. Hendrie Lloyd.

*History.*—The case was interesting because the roentgen ray was particularly accurate in locating the lesion. The patient was a man, aged 34, with a history of venereal infection, who was admitted to hospital with severe frontal headache, vomiting and dizziness. The vomiting was quite characteristically cerebral in type, that is, projectile, frequently repeated, without nausea, and without reference to food in the stomach. The giddiness was shown by a staggering gait to the left. The patient had lost one eye from accident, but the fundus of the other eye was reported normal. Unfortunately the form field was not taken, partly because the patient's mental state made

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* On March 10 the vision of O. D. was 6/5, of O. S. 6/9. The disk in each eye was level with the fundus.

cooperation difficult. The Wassermann test of the blood was positive, but for the spinal fluid it was reported negative, although there was a cell count of 40.

*Diagnosis and Treatment.*—A diagnosis was made of cerebral syphilis, with possible gumma in the right hemisphere, as indicated by a very slight left hemiplegia and astereognosis. This diagnosis was further indicated by the roentgen ray, the report being that there was a dense circumscribed shadow in the posterior cerebral region on the right side, very suggestive of a new growth, probably gumma. The localization indicated by the shadow was the parieto-occipital region. The patient was put on specific treatment, mercurial inunctions and the iodids. He did not do well. The headache and vomiting persisted, and a stuporous state came on. A decompressive operation was then done by Dr. A. C. Wood over the parieto-occipital region, as indicated by the roentgen-ray. The cortex was found bulging and rather soft, but the man's condition on the table was bad, and nothing was accomplished except decompression. The patient died on the following day.

*Necropsy Report.*—The brain shows a gummatous infiltrating meningitis about the tip of the temporal lobe, extending backward. In the interior of the parieto-occipital region is a new formation, which under the microscope is seen to be characterized by a very free lymphocyte infiltration.

DISCUSSION

Dr. Charles K. Mills said in this case which he saw with Dr. Lloyd he had made the diagnosis of a parieto-occipital lesion, probably a tumor. The man had a doubtful hemianopsia, some impairment of muscular sense and also possibly cutaneous sensibility. The patient's mental state was such that Dr. Mills could not get out all the points that he desired. The necropsy seems to have confirmed the diagnosis, except that there was more present than Dr. Mills had supposed.

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## THE CENTRAL NERVOUS SYSTEM IN PURPURA HEMORRHAGICA. Presented by Dr. Alfred Gordon.

No record appears to be in the literature concerning the intimate histologic state of the nervous tissue in cases of purpura hemorrhagica. In the case described by Dr. Gordon there was no hemorrhage on the surface of the brain or in the cerebral cavities which is found in hemophilic cases, but profound changes were seen in the tissue of the brain and spinal cord and everywhere confined exclusively to the gray matter. The patient was a child, 5 years of age. His brain and spinal cord on gross appearance were extremely pale. The tissue was very soft and appeared flattened. Nowhere could be seen a single vessel showing the presence of blood in it. Large sections of the brain revealed the same unusual pallor over the entire sectioned surfaces. The cerebral cortex histologically shows diffuse vacuolation. A very large number of cells are destroyed. Empty round spaces filled or not with blood clots or with round cells are seen over extensive areas. In some places empty spaces of the shape of blood vessels, on one side of which a row of round cells is seen. This condition was found over the entire cortex, but much more pronounced in the motor area than in any other portion. The occipital lobe was the least affected. No vacuolation was

observed in the layers of the cerebellar cortex. Basal ganglia, mid-brain, and medulla presented a similar appearance. The condition was totally absent in the white substance of the brain. In the spinal cord the pathologic state was precisely the same. The empty spaces, vacuolation, and destruction of cells were all confined to the cornea, more in the anterior than in the posterior ones.

Dr. Gordon then calls attention to contrast with anemias, in which destruction and degeneration are confined exclusively to the white substance. The involvement of the gray substance in purpura hemorrhagica could perhaps be explained by its more abundant blood supply than that of the white matter, also by the fact that the central branches are of a terminal variety, so that the destruction of tissue is rapid and there is no tendency for repair.

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SPECIAL COMMUNICATION. Presented by Dr. William G. Spiller.

Dr. Spiller spoke of a condition he had observed recently in *epidemic cerebrospinal meningitis*. The palpebral fissures were considerably enlarged, the pupils also probably were dilated. Dr. Spiller regarded this as a sign of irritation of the cervical sympathetic by the meningitis. The appearance of wild staring eyes presented by the patient was remarkable. Dr. Spiller referred to four cases which he believed were examples of *transverse myelitis caused by influenza*. In three the diagnosis seemed certain, and in one it was very probable. Two patients showed considerable improvement after a time. One patient died and the fourth doubtless also will die, as his condition is exceedingly grave.

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## CHICAGO NEUROLOGICAL SOCIETY

*Regular Monthly Meeting, Feb. 20, 1919*

Hugh T. Patrick, M.D., *President*

SO-CALLED LETHARGIC ENCEPHALITIS was discussed by Dr. Peter Bassoe.

He stated that during the past few weeks he had been very much impressed with the fact that there was in Chicago an unusual number of cases of encephalitis which followed in the wake of the influenza epidemic. Looking back through the history this was found to have occurred before. In 1745, there was an epidemic in Germany where it was noted that there were many cases of somnolence. This observation was repeated off and on and in the large epidemic of 1889 and 1890 there was a large number of cases of nervous complications, among them a good many cases which were designated as "nona"—a mysterious disease which did not seem to be anything very tangible but was described in medical journals. One Austrian writer maintained that it was an encephalitis chiefly located in the midbrain.

One of the most complete papers of that time on the nervous sequelae of influenza was written by Dr. Archibald Church in 1891. It spoke of multiple neuritis, poliomyelitis, encephalitis, and so on, but the term "nona" did not seem to have reached over here.

Nothing more was heard of "nona," but in 1917 and 1918, after the influenza epidemic, there appeared what in Austria first was called "lethargic encephalitis." An epidemic also appeared in England which first was thought to be due to food poison and then to be a peculiar poliomyelitis but this idea was discarded because there were no cases of ordinary poliomyelitis, and the affection was looked on as a new disease.

In France Sainton claimed it was a cerebral form of influenza, and both he and Wilson in England recalled the old "nona" and realized it was the same malady. The disease has been best described by S. A. K. Wilson in England and Sainton in France, and their descriptions are very similar. The symptoms were somnolence, fever, ocular paralysis, not much headache, and very few meningeal symptoms. There have been some necropsies and a simple encephalitis of the pons and midbrain was found.

Within the last few weeks there had been a number of such cases in Chicago. He referred briefly to those he had seen, asked if the other members of the Society had seen similar ones, and requested them to look out for them.

Case 1.—A girl, aged 10, on Dec. 27, 1918, showed palsy of the right external rectus. Two weeks later she developed spastic paralysis of the left arm, slight spastic weakness of the left leg. On February 1, difficulty in speech and swallowing set in. She was admitted to the hospital on February 3 and remained in an almost stationary condition for over a week. There was weakness of the facial muscles on both sides. Ankle clonus and the Babinski sign were present on the left side. On February 15, respiratory difficulty suddenly appeared and the patient died. No necropsy. Spinal fluid obtained on February 5 gave a cell count of 4, weakly positive Nonne test and negative Wassermann reaction. The Lange test was quite strong (2343321100).

Case 2.—A woman, aged 34, about January 20, gradually became listless and drowsy. When examined on February 10 she was disoriented and confused. The temperature was normal, but the pulse was rapid. There was right facial palsy, and protrusion of the tongue was very weak. The tendon reflexes in the left leg were relatively increased. During the next few days the temperature rose and reached 105.6 F. before death from respiratory failure on February 13. At the necropsy no gross brain changes were found aside from congestion which was present in all viscera. Sections from the lenticular nucleus, pons and medulla, show numerous and large perivascular collections of mononuclear cells. The condition was most marked in the upper part of the pons, where hemorrhagic areas also were seen.

Case 3.—A woman, aged 44, gradually became listless, drowsy and unsteady from about February 1. In walking the right foot was dragged. When seen on February 15, she had a mask-like facial expression and the eyes were half closed. She was conscious, but answered questions in monosyllables only. The facial muscles were weak on both sides, more so on the left. Both arms and legs were moderately rigid, more so on the right side, but the reflexes were practically normal. The spinal fluid was under normal pressure, gave a negative Wassermann test, very weak Nonne and Lange tests, and a cell count of 9. [The patient's condition remained unchanged for a time, then the extremities gradually grew more rigid and there was coarse jerking on attempted movement. Retention of urine set in on February 21. On February 23, while being fed with a spoon, she suddenly choked, developed signs of pulmonary edema and died in two hours. At the necropsy no gross

changes were found in the brain, but histologic examination revealed changes similar in nature and location to those mentioned in the previous case.]

CASE 4.—A girl, aged 13, on February 4, complained of buzzing in the ears. The eyes and throat then became red and on February 10, when the temperature rose to 102 F., she was suspected of having influenza. She became drowsy and had no headache. On February 11 the leukocyte count was 12,200. When seen on February 14, she showed signs of bilateral facial weakness and suggestion of a left Babinski sign. The deep reflexes were increased, more on the left side. She was removed to the hospital and has remained in an extremely lethargic condition. Since February 10 she had been entirely tube fed. The temperature had usually been about 101 F., and the extremities and neck had become very rigid. Repeated cultures of the blood and spinal fluid were negative. Nystagmus had been constant.

[When last seen, on March 12, the condition was practically unchanged.]

CASE 5.—A man, aged 44, was taken with diplopia on February 9. This lasted three days. On February 13 he complained of weakness in the left hand and the temperature reached 103 F. On February 18, retention of urine, weakness and stiffness of the jaw and stiffness of the neck appeared, together with marked listlessness and somnolence. He was admitted to the hospital on February 20. [The condition gradually grew worse, with increasing rigidity of all the muscles of the body. A coarse tremor developed whenever voluntary movement was attempted. The patient never could protrude the tongue beyond the lips, but speech and swallowing remained fairly good. The temperature gradually rose, but the patient remained conscious until a few hours before death on March 8. No organisms were obtained in cultures from blood and spinal fluid; the latter showed increase in cells and globulin. No necropsy.]

The speaker called attention to the preponderatingly extrapyramidal character of the motor disturbance in these cases, the condition of the muscles and the facial expression bearing considerable resemblance to that of paralysis agitans. This condition was ascribed to lesions in the basal ganglia. These cases will be reported in greater detail in the near future.

### DISCUSSION

DR. SIDNEY D. WILGUS said he had seen a similar case a short time before. His patient was a man who suffered with influenza about December 1, but this passed off uneventfully in a few days. Then followed several weeks of lassitude. Early in January he began to sleep a great deal, and about this time he noticed that he saw double. The diplopia lasted not more than a week and then disappeared, and has failed to recur. When seen by the family physician at this time his pulse was about 50 and he felt rather weak, but complained of little else except neuralgic pains in the occipital region. Some ten days after the diplopia disappeared he was suddenly stricken with a right facial paralysis. At the same time his tongue was protruded distinctly to the left. There were disturbances of taste. His temperature by this time had been running from 99 to 102 F., and perhaps averaged 101 in the afternoon. There was a distinct Gordon reflex on the left, but there were no other abnormalities of the reflexes. Later this patient developed double facial palsy and quite marked difficulty in swallowing. He appears to be a very sick man. This case had not been thought of in connection with influenza, but had been watched with a good deal of interest; in view of the discussion by Dr. Bassoe, it can be placed quite clearly under the heading of influenzal encephalitis.

Dr. L. Harrison Mettler asked if these cases were always preceded by the ordinary type of bronchial influenza.

Dr. Bassoe replied that this was not always the case, but the cases occurred in localities where influenza had been prevalent.

Dr. Bayard Holmes said he saw a somewhat similar case a few days before, but the patient did not have the eye symptoms and was not drowsy. He thought it was a case of paralysis.

Dr. Hugh T. Patrick stated that he had seen one case of the basal ganglia type and one other that might have belonged to this group, but he had been inclined to think it a case of poliomyelitis of the cerebral type.

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## HISTOPATHOLOGY OF CARCINOMA OF THE CEREBRAL MEMBRANES. Presented by Dr. George B. Hassin.

Dr. Hassin read a paper with this title and demonstrated the specimens. (Printed in full in this issue.)

### DISCUSSION

Dr. Peter Bassoe asked if there was any involvement of the skull and if the skull cap had been examined, as it was common for carcinoma of the breast to have bone metastases.

It reminded him of a case of endothelioma of the dura in which there was a crater-shaped depression of the dura in the occipital region. The tumor spread on the dura in all directions and had grown into and completely filled the superior longitudnal sinus. It had also grown to and almost filled the occipital lobe. Into this crater-shaped depression there fitted a thick button of bone, which, when decalcified, was found infiltrated with tumor.

Dr. Bassoe thought he had once shown the society metastatic carcinoma of the brain from a breast case. Two thirds of the right hemisphere was involved by the carcinoma. There were no pressure symptoms, but very profound aphasia and almost complete loss of mind. The brain tissue was replaced by carcinoma without any material increase in volume.

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## CASE WITH SYMPTOMS OF DERCUM'S DISEASE AND HYSTERICAL MANIFESTATIONS. Presented by Dr. Samuel N. Clark.

*Abstract.*—The symptoms of Dercum's disease in the case reported were as follows: General adiposity, as indicated by a weight of 188 pounds in a woman 4½ feet in height; lipomata, about 120 in number ranging in size from that of a hazelnut to a chestnut distributed subcutaneously on arms, forearms, buttocks and thighs, accompanied by an itching sensation, but practically without pain. At 33 years of age, following an illness, presumably pleurisy, the patient had frequent spells of coughing, apparently voluntary in character, followed by expectoration of bright red blood in amounts described as about as much as a teacupful. Examination of the chest and of the sputum showed no evidence of disease. Finally, the situation became so "deplorable" that the pleural cavity was exposed. No abnormal condition was found. At about 38 years, coincidentally with the disappearance of hysterical manifesta-

tions, the hemorrhages ceased. It is believed that the tendency to hemorrhages, which is a feature of Dercum's disease, coupled with a hysterical type of reaction, gave rise to the hemoptysis.

At 33 years, following the death of a lover, the patient developed fixed phantasies of marriage and motherhood with occasional tantrums, hysterical paralyses and other hysterical conversions. Commitment was precipitated at 37 by alleged attempt at suicide. The phantasies and hysterical conversions disappeared during the year and a half following commitment. A parallel is drawn between the phantasies and an alleged acute illness during which it was demonstrated that the patient caused the thermometer to register falsely. Here, as in the case of the phantasies, the obstacles to the end desired were swept aside by repression and pretense. Following the exposé of the thermometer incident, all complaints referring to the illness ceased abruptly because they were rendered untenable by the fact that the pretense of fever was clearly exposed. The phantasies, tantrums, etc., likewise became untenable in the face of a combination of factors, among which may be mentioned an apparent lack of attention of the physician to the hysterical manifestations, definite proof furnished that certain beliefs could not be true and the knowledge that one in whom she gradually had been educated to have confidence did not share her belief in the phantasies. This view lays stress on the type of reaction rather than on repressed complexes in the genesis of the phantasies. The significance of the view in relation to psychotherapy is manifest.

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RECOVERY IN PARANOIA. Presented by Dr. Richard Dewey.

Dr. Dewey read a paper on this subject.

# Book Reviews

A REVIEW OF NERVE INJURIES AND THEIR TREATMENT. Sir James Purves Stewart, K.C.M.G., C.B., M.D. (Edin.), F.R.C.P., and Arthur Evans, M.S., M.D. (Lond.), F.R.C.S. Second Edition. Oxford University Press, London, 1919.

This is the second edition largely rewritten and revised by the authors, both of whom have had large war experience. This war, neurologically speaking, has furnished exceptional experience in injuries of the peripheral nerves and in the neuroses. There have been a number of books on peripheral nerves. There is no one book, however, which gives as much information and presents the subject as well as this small volume, for it not only gives in a brief and succinct manner the neurological and surgical aspects of the subject, but it also gives to the beginner an adequate presentation, so that for example, a ward surgeon or a young neurologist can get out of this volume all the information that he needs to enable him to examine, chart and digest injuries of the peripheral nerves.

The authors still adhere to the idea that regeneration takes place both from the peripheral, as well as the central end of a nerve. They accept and apparently their own experience confirms the sensory views of Head. This question has not been definitely solved. There is a great deal of work in this field being done by American physicians in the various army hospitals, designated for this purpose. Fortunately a peripheral nerve commission has been appointed by the Surgeon-General and no doubt when this work is completed and published, it will be a credit to the American profession, as well as to the Surgeon-General's Office.

The authors' definition of reflex paralysis is interesting. To quote from them: "This condition (originally described by Babinski and Froment) appears to be due to a functional (but not psychogenic) hyperexcitability of the anterior cornual cells in the corresponding segment of the spinal cord, in cases where the reflex paralysis is of a spastic type; or a functional (but not psychogenic) inhibition of hypothermia and cyanosis are doubtless due to disorder of the adjacent nucleus sympatheticus in the anterolateral cornu of the cord, which controls the blood vessels of the limb." There are very few examples of this condition in the American Army and it is the opinion of most neurologists who have seen these patients, that they are functional in character, disagreeinng in their view from those held by the authors, and by Babinski and Froment.

The authors give statistics of 520 cases of nerve injuries. There is nothing unusual in the distribution of the nerves injured, they being much like those furnished by other authors, with the exception that in the upper limbs, the ulnar nerve was injured more than the musculospiral. There is an unusual number of median nerve cases—forty-four. The illustrations are first rate. The discussion of the surgical treatment is quite up-to-date.

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